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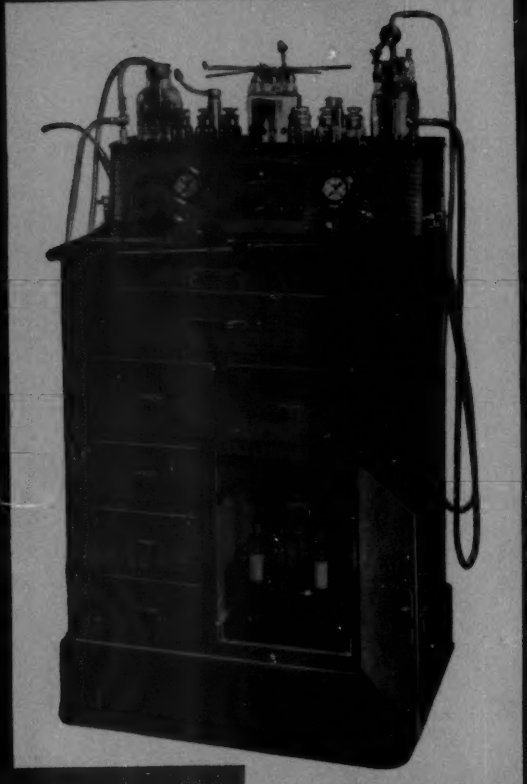

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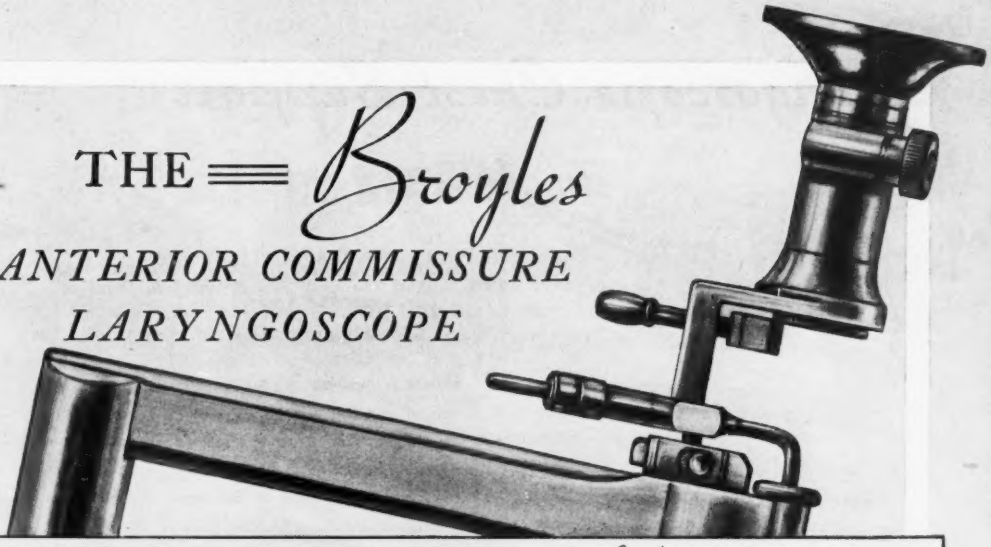

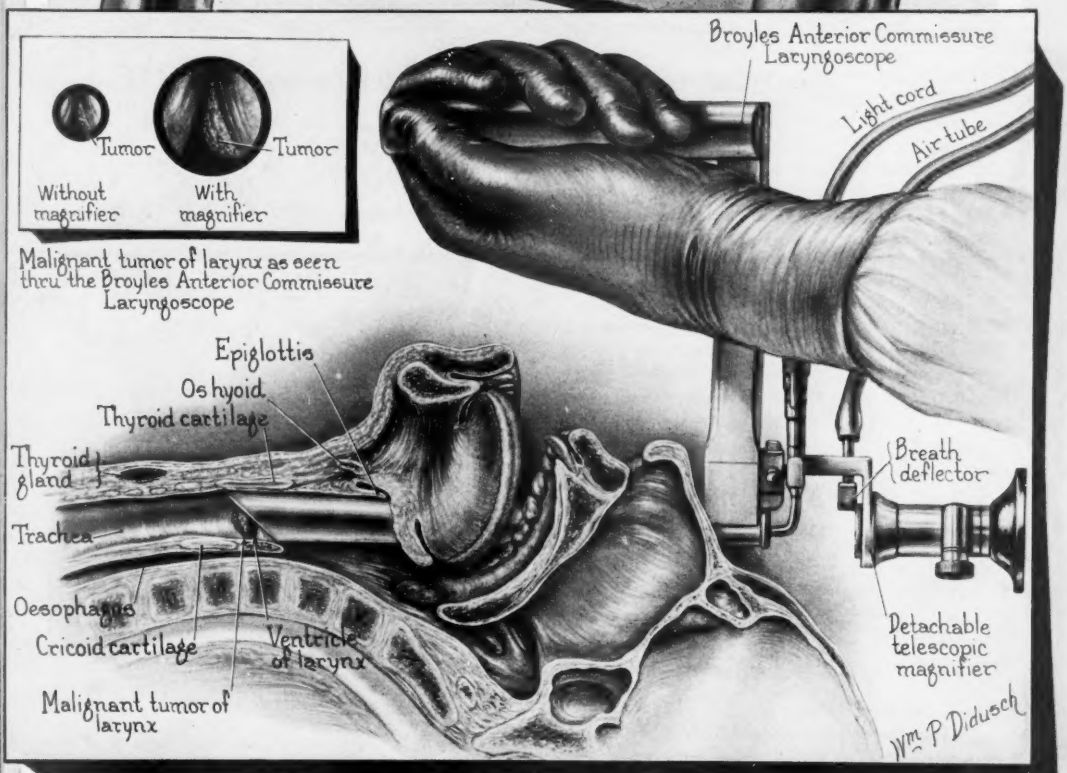

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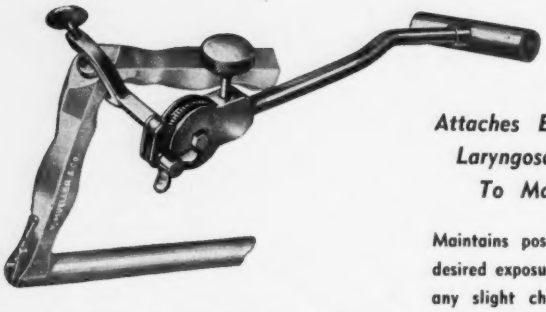

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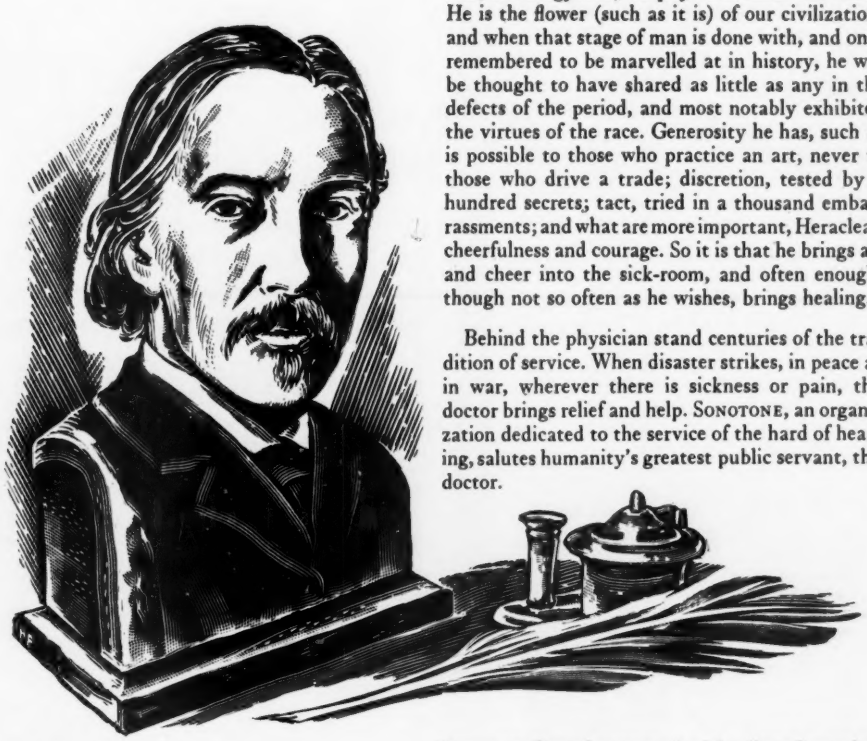

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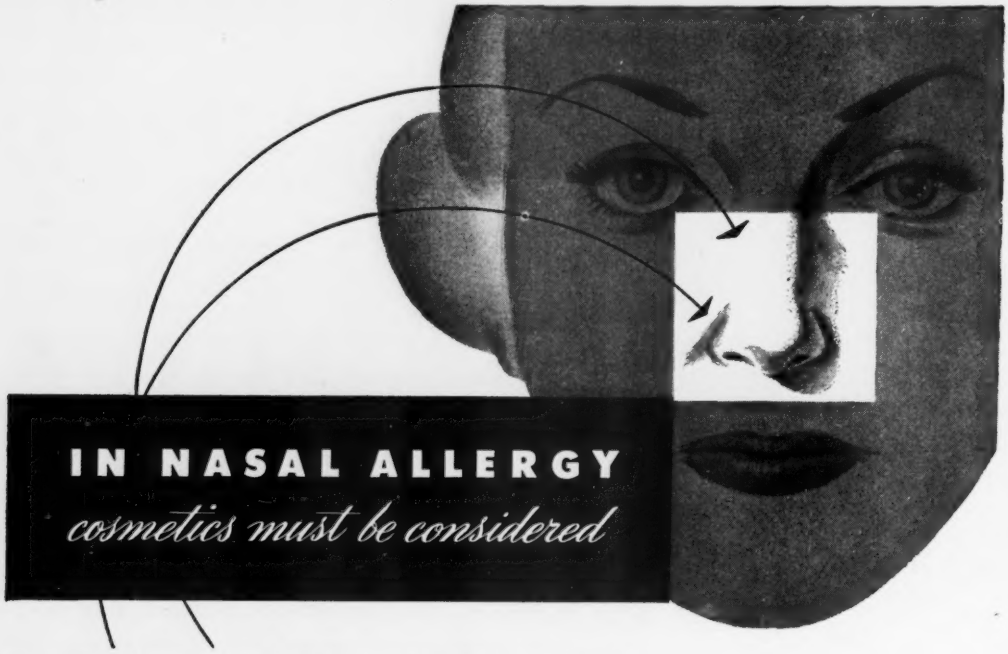

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ETIOLOGIC ANALYSIS OF ONE HUNDRED CASES OF MÉNIÈRE'S SYMPTOM-COMPLEX

LAWRENCE K. GUNDRUM, M.D.
LOS ANGELES

IF THE cause of a symptom or of a disease can be determined, the choice of treatment is, of course, simplified. Cytologic examination of the postnasal secretion in patients with Ménière's symptom-complex has been invaluable in my experience in diagnosis of the underlying cause. It has facilitated choice of therapy.

I realize that many of the 100 cases I am about to discuss probably are not instances of true Ménière's disease, but all patients exhibited the symptom-complex of Ménière's syndrome.

The Figure is a smear of the postnasal secretion from a man of 45 who had typical Ménière's syndrome. He had been unable to get up, was in bed in the hospital, and was unable to walk. I asked that a specimen of the postnasal secretion be examined; the hospital laboratory reported there were no eosinophiles in the secretion. Nothing of a pathologic nature could be discovered; neither was the history or the findings suggestive of allergy. The patient was discharged from the hospital, and the next day was brought to my office. He had typical vestibular findings, nerve deafness, and vomiting. I took a smear and found pure eosinophilia. On antihistaminic therapy he promptly recovered; all symptoms disappeared. Unfortunately, he did not return to the office after recovery, and so I was unable to test his hearing following treatment.

This discrepancy in the results of the test for eosinophiles in postnasal secretions emphasizes the importance of cytologic study of such materials by the physician responsible for diagnosis. At the hospital the secretion was negative for eosinophiles. The Figure is from the specimen I obtained at the office within a day after the specimen which was taken and examined at the hospital was reported as negative for eosinophiles.

The series reported here consists of 100 consecutive cases of so-called Ménière's syndrome, or labyrinthine hydrops, from my own practice. The data presented chiefly concern the observation of eosinophiles or neutrophiles or of the absence of these cells in the postnasal secretions of the patients so afflicted and the clinical results of therapy based on the cytologic picture of the secretions.

Because of referral to me of many industrial accident cases, I probably have seen more than the average number of patients who exhibit symptoms typical of Ménière's symptom-complex apparently brought about by head injury. Forty-three of the one hundred cases were of this nature; the cytologic pattern of the postnasal secretion was normal in all but three cases, in each of which there was neutrophilia.

Presented at the Western Colorado Spring Clinic, Grand Junction, Colo., April 19, 1952.

The remainder of the series, 57 cases, I have subdivided as 34 cases associated with allergy, 10 associated with infection, and 13 in which the underlying cause of the Ménière's symptom-complex could not be attributed to allergy, infection, or injury.

The subjective data on which the diagnosis of Ménière's symptom-complex was based in the entire series of cases consisted in dizziness, spontaneous nystagmus,

Smear of the postnasal secretion from a man with typical Ménière syndrome.

TABLE 1.—*Comparison of Subjective Data in Ménière's Symptom-Complex in Relation to Probable Cause*

	Allergy (34 Cases), %	Infection (10 Cases), %	Injury (43 Cases), %
Dizziness	100.0	100.0	90.6
Staggering	97.0	100.0	83.7
Nausea	92.1	90.0	34.9
Tinnitus	55.9	20.0	65.1
Deafness	58.8	40.0	90.6
Allergic symptoms and/or history	35.3	10.0	0
Sinusitis symptoms	17.6	10.0	4.6
True vertigo	32.3	30.0	46.5
Headache	23.5	10.0	37.2

staggering, nausea, tinnitus, deafness, allergic symptoms and/or history, symptoms of sinusitis, true vertigo, and headache.

Table 1 lists the incidence of subjective symptoms recorded in the groups associated with allergy, infection, or injury.

Examination for the presence of spontaneous and positional nystagmus, type and degree of deafness, vestibular function, and cytologic picture of the postnasal secretion was done also. The diagnosis of nerve deafness in this series was based on the hearing curve and on the phenomenon of recruitment.

The subjective data alone give but few clues as to the underlying cause of the disease.

Dizziness was present in almost all patients regardless of probable cause of the Ménière symptom-complex.

Staggering was exhibited by almost all in the allergy and infection groups, but was noted in only 83.7% of the injury group.

The injury group is distinguished also by the relatively low incidence—34.9% —of nausea, whereas approximately all patients in the allergy and infection groups complained of this symptom.

Tinnitus was present in more than half of the patients in the allergy and injury groups but in only 2 of the 10 patients in the infection group.

The complaint of deafness stands out prominently in the injury group—all but 4 of the 43 had deafness; on test, nerve deafness was predominant. Deafness was

TABLE 2.—*Comparison of Objective Data in Ménière's Symptom-Complex in Relation to Probable Cause*

	Allergy (34 Cases), %	Infection (10 Cases), %	Injury (43 Cases), %
Spontaneous nystagmus	23.5	30.0	39.4
Positional nystagmus	5.8	0	13.9
Deafness			
Nerve	32.3	30.0	83.7
Mixed	17.6	40.0	2.32
Complete	0	0	2.32
Conduction	11.9	0	2.32
Response to vestibular tests			
Hyperactive	26.4	10.0	41.8
Hypoactive	11.9	0	2.32
Hyperactive-hypoactive	0	0	2.32
Cytologic picture of postnasal secretion			
Eosinophiles	82.3	0	0
Neutrophiles	5.8*	100.0	6.9
Normal	11.9*	0	93.1

* Included in allergy group in view of history and/or symptoms of allergy.

complained of by 58.8% of the patients in the allergy group and by 40% of those in the infection group.

Allergic symptoms and/or history played but a small part in the symptomatic picture. Only 12 of the 34 patients in the allergic group reported this and only 1 of the 10 in the infection group. None of those in the injury group gave such a history.

Sinusitis symptoms presented a fairly similar proportion.

True vertigo appeared in 46.5% of the patients in the injury group and in 32.3% and 30% respectively of those in the allergy and infection groups.

Headache occurred in 23.5% of the patients of the allergy group, in 10% of those in the infection group, and in 37.2% of those who had suffered head injury.

The objective data are given in Table 2. Differences in the three groups are not remarkable with the exception of the predominance of nerve deafness and the constitutional response to douching in the injury group. For example, after douching for one minute with water 68 F., sweating, pallor, and nausea with violent vomiting appeared in patients in the injury group. Such a response to douching did not occur in the allergy and infection groups. The cytologic picture, of

course, is of significance. In the infection group, although 40% of the patients complained of deafness, on auditory test 70% were found to have some degree of loss of hearing.

MÉNIÈRE'S SYMPTOM-COMPLEX ASSOCIATED WITH ALLERGY

In 28 of the 34 cases in whom the clinical picture of Ménière's symptom-complex was attributed to allergic factors, eosinophiles were observed in the postnasal secretion. Two other cases were included in this group because of the very definite history and symptoms of allergy, although the postnasal secretions of the patients contained only neutrophiles. The postnasal secretions of four other patients were normal, but in their cases also the history and symptomatology was such as to suggest an allergic factor.

The importance of the cytologic examination of the postnasal secretion in Ménière's symptom-complex is demonstrated particularly in this group. Only 12 patients (35.3%) had any allergic symptoms or history of allergy, either personal or familial. Had cytologic study of the postnasal secretions not been done, the cause of the symptoms in 16 (47%) of this group might have been missed. Twenty-seven of these 34 patients received allergic therapy. In 18 this consisted in standard cutaneous testing and desensitization, and in 9 antihistaminic therapy was administered. One patient received antibiotics. Six of the 34 did not receive treatment of any kind.

Of the 27 patients who received allergic therapy, 26 were improved symptomatically. The hearing in 11 of this group was normal initially. Sixteen had impaired hearing prior to treatment for their allergies. Test of hearing after treatment revealed improvement in seven, and no change in one; in another the hearing had become worse. Seven of the patients with impaired hearing before treatment failed to return to the office for test of this function.

MÉNIÈRE'S SYMPTOM-COMPLEX ASSOCIATED WITH INFECTION

In all 10 cases of the group in which the Ménière symptom-complex was associated with infection, neutrophiles were observed in the postnasal secretion. Treatment in eight consisted in administration of antibiotics or histamine and use of a low-sodium diet. Six of the eight patients obtained symptomatic relief following treatment. In one case the hearing was improved; in 3 it was unchanged, and in 3 others an auditory examination after treatment was not made. One patient received, in addition to the histamine and a low-sodium diet, radium therapy to the postnasal space. This was followed by good results in hearing, but it did not produce symptomatic relief. One patient did not return for treatment.

MÉNIÈRE'S SYMPTOM-COMPLEX NOT ASSOCIATED WITH ALLERGY, INFECTION, OR INJURY

The cytologic picture of the postnasal secretion was normal in nine cases of Ménière's symptom-complex not associated with allergy, infection, or injury. In 3 of these cases the patients were discovered to have central nervous system lesions; in the other six treatment consisted in use of histamine, a low-sodium diet, and nicotinic acid. Four patients received symptomatic relief, and one had improved hearing after treatment.

Four patients with Ménière's symptom-complex were not tested as to cytologic content of the postnasal secretion. Two did not return for treatment. One received

histamine and was put on a low-sodium diet, which did not effect improvement in either the symptoms or the hearing. The other patient received histamine and antibiotics and was put on a low-sodium diet; good results were obtained symptomatically; there was no loss of hearing in this patient initially.

I shall present two cases in detail.

CASE 1.—Mrs. J. S., a woman aged 56, was first seen by me in December, 1948. Her personal and familial history were irrelevant except that she stated that she had chronic sinusitis. There was nothing in her history to suggest allergy. She had had an attack of vertigo six months before, had fallen, and was of the opinion that she had been unconscious for a few seconds. On regaining consciousness she had some pain in her face and in the occipital region. She was unable to walk without assistance. Since the initial attack she had had frequent repetitions with nausea and vomiting and what she described as nystagmus. She was never free from slight dizziness and was forced to use a cane when walking. Tinnitus was continuous.

Physical examination, including complete neurologic and serologic tests for syphilis, disclosed nothing abnormal or pathologic.

The patient had a deviated nasal septum. On examination with the nasopharyngoscope the posterior lip of the left Eustachian tube was observed to fail to move on swallowing. A slight loss of hearing for the tones above 4096 cps was noted. Tests of the vestibular apparatus revealed poor responses in both ears. She was returned to the care of her family physician with the usual suggestions as to therapy—that is, use of histamine, low-sodium diet, and barbiturates.

A year and a half later she returned stating that the dizzy spells were much worse and were almost constant, and that she had spent two months at a widely known clinic and had been told that there was no treatment for her condition. She was unable to drive a car or to walk without assistance.

Examination at this time, August, 1949, gave essentially negative results except that hearing was much worse, having dropped to an average of 40 db. in the left ear and 50 db. in the right. Material obtained from the postnasal space was loaded with eosinophiles. She was given extensive intracutaneous tests and exhibited severe reactions to many pollens and other allergens.

Use of a desensitizing antigen was started on Sept. 27, 1949. In January, 1950, she was able to walk without the use of a cane and was driving her own car; the dizziness, while present occasionally, was only slight. Her hearing was considerably improved but was not quite so good as it was at the time I originally examined her, in December, 1948. The most recent report from this patient—a few weeks ago—indicates no return of the Ménière symptom-complex.

CASE 2.—A. F., a man aged 38, first came to my office in January, 1949. He complained of severe pain in the left side of his face. Pus was seen in the left nostril. The left maxillary sinus was irrigated through the natural ostium, and a large amount of purulent material was obtained. The condition apparently subsided.

He was next seen Aug. 23, 1949, at which time he complained of deafness, staggering, and true vertigo. Secretion from the postnasal space demonstrated eosinophiles. Auditory examination revealed mixed deafness of both ears, averaging a loss of 30 db. in each. Tests showed the vestibular apparatus normal except that no response was elicited to douching of the left ear with either hot or cold water. Neurologic examination did not reveal any pathologic condition. On intracutaneous tests there were severe reactions to pollens and other allergens.

Allergic treatment was started Sept. 15, 1949. The patient has shown a gradual improvement. On Oct. 19, there was response to douching of the left ear with water at 68 F. after two and one-half minutes. At present the patient is symptom-free, and the hearing has improved about 10 db. in each ear.

Improvement in these patients with Ménière's syndrome has been gauged almost entirely on purely symptomatic evidence, but in the case just described the objective finding of return of the nystagmus on douching proved beyond any doubt that the condition had responded both subjectively and objectively to the allergic treatment.

COMMENT

In so small a series of cases, of course, definite conclusions as to the etiologic significance of allergy and infection in Ménière's symptom-complex cannot be drawn. The observations suggesting allergy in the 34 cases so classified do not prove that allergy was the sole cause of the vestibular symptoms. However, the relief of dizziness, staggering, tinnitus, vertigo, and other manifestations of vestibular disturbance following allergic therapy, as well as the improvement in hearing following treatment for their allergies in the great majority of the patients in whom this deficiency had been observed, would seem to offer a measure of confirmation that the Ménière syndrome as exhibited by the patients in this particular group of cases could be attributed to allergy. Likewise, the relief of the vestibular symptoms characteristic of Ménière's disease by treatment aimed at combating infection would suggest that this syndrome in some instances can be caused by infectious processes.

The diagnostic accuracy of cytologic examination of postnasal secretions is confirmed in this series. Sixteen patients who had neither a history—personal or familial—nor symptoms of allergy had eosinophiles in the postnasal secretion. Had this test not been made, the allergic factor as a probable cause of the Ménière symptom-complex would have been missed. Thirteen of these 16 patients received allergic therapy, and all 13 were relieved of their vestibular symptoms, while the hearing became improved in 4 who had had impaired hearing prior to the allergic therapy. In the patients whose Ménière syndrome was associated with a neutrophilia of the postnasal secretion, this cytologic observation was of value in pointing to the necessity for antibiotic therapy in addition to the usual treatment for Ménière's symptom-complex.

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HEARING LOSSES AFTER EXPERIMENTAL LESIONS IN BASAL COIL OF COCHLEA

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AND
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PAST INVESTIGATIONS have shown that the auditory spectrum has a spatial representation within the cochlea.[1] The low frequencies have their most effective action on the structures in the apical turn and the high frequencies on those structures in the basal turn of the cochlea.

Although these general facts are well known, there is incomplete information regarding the effect of frequency and intensity on the location and spread of sensory and neural excitation within the cochlea.

One method of studying the problem is to produce restricted lesions in the cochlea and to determine the effect on auditory function. The method also provides information on the pathology of perception deafness. We have performed a series of such experiments on cats which were conditioned for auditory testing.

From the Division of Otolaryngology of the University of Chicago.

This project was supported by a grant from the Veterans Administration, contract No. V-1001-M-2354.

1. Some of the more representative references are given without an attempt to list them all. Békésy, G. V.: Variation of Phase Along the Basilar Membrane with Sinusoidal Vibrations, J. Acoustical Soc. America **19**:452-460, 1947; On the Resonance Curve and the Decay Period at Various Points on the Cochlear Partition, ibid. **21**:245-254, 1949. Culler, E. A.; Coakley, J. D.; Lowy, K., and Gross, N.: A Revised Frequency Map of the Guinea-Pig Cochlea, Am. J. Psychol. **56**:475-500, 1943. Davis, H.; Lurie, M. H., and Stevens, S. S.: Symposium: Is There Localization in the Cochlea for Low Tones? Ann. Otol. Rhin. & Laryng. **44**:776-777, 1935. Galambos, R., and Davis, H.: The Response of Single Auditory-Nerve Fibers to Acoustic Stimulation, J. Neurophysiol. **6**:39-58, 1943. Gross, N. B.: The Effects of Cochlear Lesions on the Auditory Response of the Guinea Pig, J. Comp. & Physiol. Psychol. **45**:127-139, 1952. Lurie, M. H.; Davis, H., and Hawkins, J. E., Jr.: Acoustic Trauma of the Organ of Corti in the Guinea Pig, Laryngoscope **54**:375-386, 1944. Schuknecht, H. F.: Deafness Following Blows to the Head: A Clinical and Experimental Study, Tr. Am. Acad. Ophth., pp. 407-417, 1951. Smith, K. R.: The Problem of Stimulation Deafness: Histological Changes in the Cochlea as a Function of Tonal Frequency, J. Exper. Psychol. **37**:304-317, 1947. Smith, K. R., and Weaver, E. G.: The Problem of Stimulation Deafness: The Functional and Histological Effects of a High Frequency Stimulus, ibid. **39**:238-241, 1949. Walzl, E. M., and Bordley, J. E.: The Effect of Small Lesions of the Organ of Corti on Cochlear Potentials, Am. J. Physiol. **135**:351-360, 1942. Wever, E. G., and Smith, K. R.: The Problem of Stimulation Deafness: Cochlear Impairment as a Function of Tonal Frequency, J. Exper. Psychol. **34**:239-245, 1944. Wever, E. G., and Neff, W. D.: A Further Study of the Effects of Partial Section of the Auditory Nerve, J. Comp. & Physiol. Psychol. **40**:217-226, 1947. Wever, E. G., and Lawrence, M.: The Patterns of Response in the Cochlea, J. Acoustical Soc. America **21**:127-134, 1949. Wever, E. G.: Theory of Hearing, New York, John Wiley & Sons, Inc., 1949.

In one series of animals [2] a lesion was made in the apex of the cochlea by introducing a small needle through the bony wall. After three months hearing tests were made, and the animals were sacrificed. It was found that lesions involving the apical turn caused hearing losses of 20 to 25 db. for frequencies of 500 cps and below. In none of the animals was there complete loss of hearing for low frequencies, in spite of the presence of severe pathological changes in the apical turn. The experiment revealed, however, that receptors in the apical turn must be present for hearing at intensities near normal threshold. The findings from one of these animals are shown in Figure 1 (Cat 1). Although there was a severe injury involving the apical 6 mm. of the cochlea, hearing for the low frequencies was depressed only about 35 db.

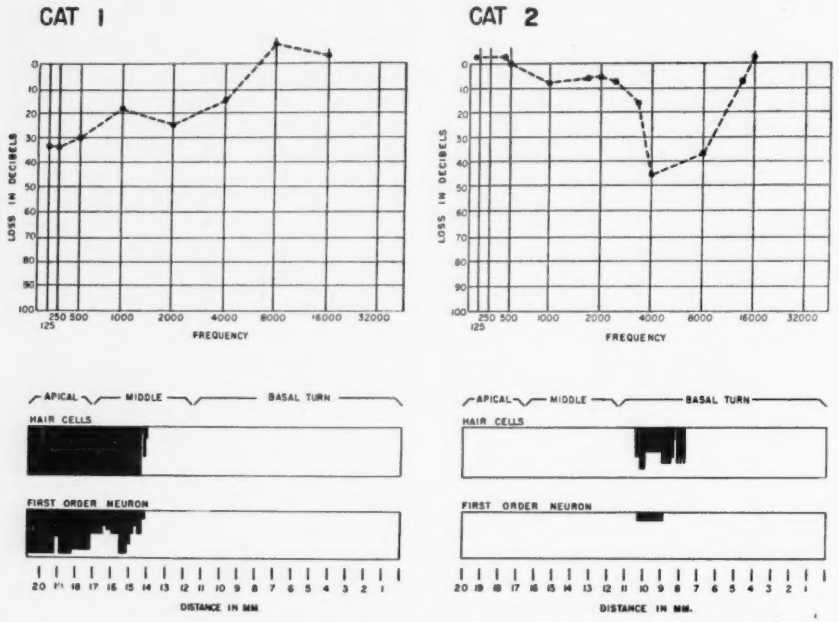

Fig. 1.—*Cat 1:* Audiogram and cochlear chart of an animal in which an experimental lesion was made in the apex of the cochlea. The method used to develop the graphs is explained in the text. The animal was sacrificed three months after the injury. *Cat 2:* Audiogram and cochlear chart of an animal subjected to a blow to the head, sacrificed six weeks after the injury.

Thus, in this ear the low frequencies at an intensity of 35 db. above threshold apparently were able to excite receptors located 6 mm. or more from the apex.

Cochlear lesions were produced in another group of conditioned cats by delivering blows to the head.[3] After several weeks hearing tests were made, and the animals were sacrificed. Histological examination of these ears showed the most marked pathological changes to be located near the middle of the cochlea (10 to

2. Schuknecht, H. F., and Neff, W. D.: Hearing Losses After Lesions in the Apex of the Cochlea, Acta oto-laryng. **42**:263-274, 1952.

3. Schuknecht, H. F.; Neff, W. D., and Perlman, H. B.: An Experimental Study of Auditory Damage Following Blows to the Head, Ann. Otol. Rhin. & Laryng. **60**:273-289, 1951.

11 mm. from the basal end) and in several animals to be restricted to this area. For example, in one animal a partial loss of sensory cells in the 8 to 11 mm. area from the basal end produced hearing losses of 45 and 35 db., respectively, for the frequencies 4000 and 8000 cps (Fig. 1, Cat 2). A consideration of the 4000 cps frequency in a number of animals showed that it selectively excited receptors located about midway between the two ends of the cochlear dust. Furthermore, it appeared that at intensities up to 45 db. for the 4000 cps frequency the sensory excitation remained restricted within a 3 mm. area.

Having determined in some degree the effect on hearing of lesions in the apical and middle turns of the cochlea, it is expedient that we turn to a study of lesions of the basal coil. An outstanding contribution on this subject was made, in 1934, by Crowe, Guild, and Polvogt.[4] They correlated the clinical and pathological findings in 79 human ears whose audiograms showed impairment of hearing limited to the high tones. The audiograms were divided into two groups, depending upon whether the loss for high tones was abrupt or gradual. In the ears with abrupt losses the most prominent lesion was atrophy of the organ of Corti and nerve in the basal coil. In the ears with gradual high-tone loss the most striking pathological condition was partial atrophy of the nerve supplying the basal turn. The study indicated that for the human the area most important for hearing at the 1024 cps frequency was more than 12 mm.; at 2048 cps, more than 11 mm.; at 4096, less than 9 mm., and at 8192, less than 5.5 mm. from the basal end of the cochlea. In approximately one-fourth of the ears no lesion was found in either the inner or the middle ear to explain the deafness.

Our approach to the problem was that of producing lesions in the basal coil of conditioned cats and of correlating the pathological findings with the hearing loss. One advantage of this method over the study of human material is that the size and location of the inner ear lesions could be controlled to some degree. Artifacts arising from postmortem degeneration of the inner ear also are avoided in work with experimental animals.

PROCEDURE

For each of six cats the function of one ear was destroyed by macerating the labyrinth with a probe introduced through the round window. The animals were then trained to respond to pure-tone auditory stimuli by moving forward in a rotating cage to avoid shock. Audiograms were determined, using this conditioned avoidance response as an indicator of hearing. The method is described in detail elsewhere.[5]

Several audiograms were made for each animal, after which an operation was performed to create a lesion in the basal coil of the experimental ear. Intraperitoneal pentobarbital (nembutal®) sodium supplemented by intravenous thiopental (pentothal®) sodium, when necessary, was used for anesthesia in all cases.

A ventral paramedian incision in the neck was made, and the auditory bulla was exposed. A cap of bone was removed in one piece so as to expose the interior of

4. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness, Bull. Johns Hopkins Hosp. **54**:315-379, 1934.

5. Culler, E. A.; Finch, G.; Girden, E., and Brogden, W. J.: Measurements of Acuity by the Conditioned Response Technique, J. Gen. Psychol. **12**:223-227, 1935. Neff, W. D.: The Effects of Partial Section of the Auditory Nerve, J. Comp. & Physiol. Psychol. **40**:203-215, 1947.

the bulla. A no. 16 bead needle was placed in a pin vise and introduced through the round window membrane into a selected region of the basilar membrane or the osseous spiral lamina. The location and the depth to which the needle was introduced were varied in different animals (Fig. 4). As fluid flowed freely from the perforation in the round window membrane, the hole was immediately sealed with a small piece of absorbable gelatin sponge U. S. P. (gelfoam®). The bony cap of the bulla wall was sutured into place and the skin incision closed. Penicillin was administered in doses adequate to maintain an effective blood level for a period of five days following surgery. The operation produced vestibular signs only in Cat 3. A horizontal nystagmus with the quick component toward the opposite side began

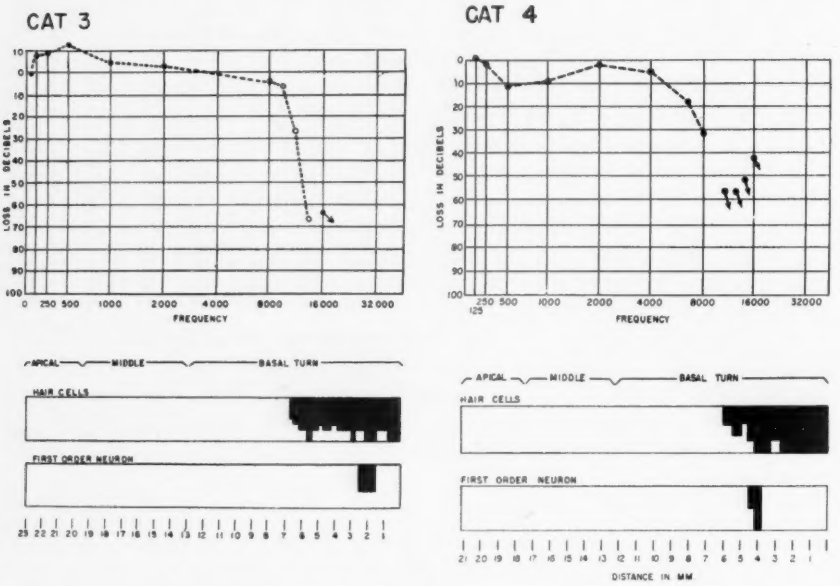

Fig. 2.—*Cat 3:* Final hearing test and cochlear chart. A needle was introduced into the cochlea 2 mm. from its basal end; the animal was sacrificed 10 weeks later. *Cat 4:* Audiogram and cochlear chart of an animal sustaining needle injury 4 mm. from the basal end of the cochlea, sacrificed five weeks after injury.

48 hours after surgery and persisted for two days. Subsequent rotation tests, however, showed normal vestibular function in that ear.

Beginning three weeks after operation, tests of hearing were made at intervals of three to seven days for each animal. The postoperative audiogram was established by averaging the results of several tests taken after thresholds were stable. After periods varying from 4 to 10 weeks following surgery the animals were anesthetized and subjected to a cortical test of auditory function. The method and the results of these cortical tests are reported elsewhere.[6] After the cortical test the animals were perfused with Heidenhain-susa solution, Helly's fluid, or 10% formalin in

6. Hind, J. E., and Schuknecht, H. F.: A Cortical Test of Auditory Function in Experimentally Deafened Cats, to be published.

isotonic sodium chloride solution. The experimental ears were removed and prepared for histological examination. The cochleas were sectioned serially; every fifth section was stained and mounted, and graphic reconstructions were made to record the pathological findings.[7]

Audiograms and charts of cochlear pathologic changes for the six cats appear in Figures 2, 3, and 5. These graphs require some clarification as to the method of development.

Hearing was tested in octave steps from 62.5 to 16000 cps, the range being determined by the testing equipment. In some animals additional frequencies were tested to determine more exactly the departure of the hearing curve from normal. Each frequency is plotted as the change in decibels between the averaged preopera-

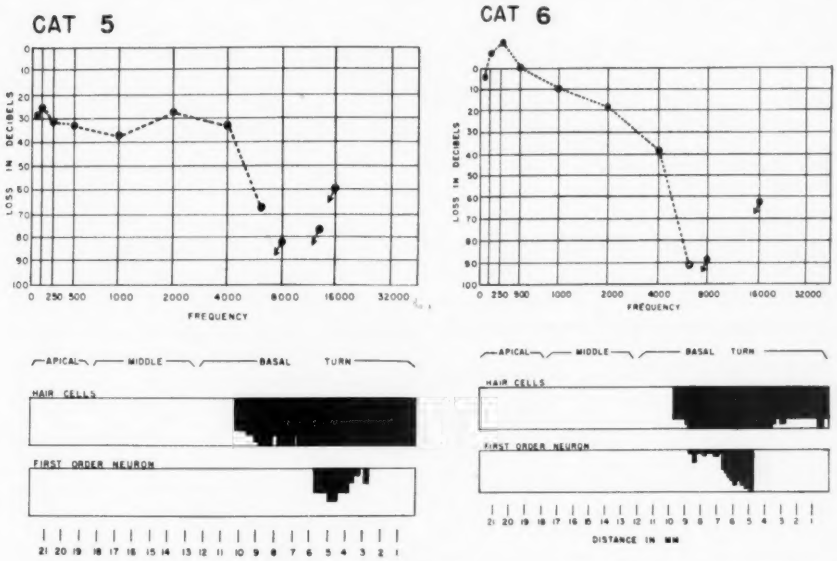

Fig. 3.—*Cat 5:* Audiogram and cochlear chart. The cat was sacrificed six weeks after a needle was introduced into the basal end of the cochlea. *Cat 6:* Hearing test and cochlear chart 5½ weeks after lesion was made in the basal coil. The spiral graphic reconstruction for this ear is seen in Figure 6, and in Figures 7 and 8 are photomicrographs showing the pathological states.

tive and postoperative thresholds for that animal; thus, a point above the zero line indicates a gain and a point below this line a loss in auditory acuity. In some animals the postoperative tests included frequencies for which no preoperative tests had been made. These measurements are indicated by a hollow circle and represent the deviation of the animal's postoperative threshold from the average normal thresholds for healthy adult cats at this frequency. The arrows in the audiograms mean that the animals did not respond to those frequencies at the maximum intensities available for testing.

7. Guild, S. R.: A Graphic Reconstruction Method for the Study of the Organ of Corti, Anat. Rec. **22**:141-157, 1921.

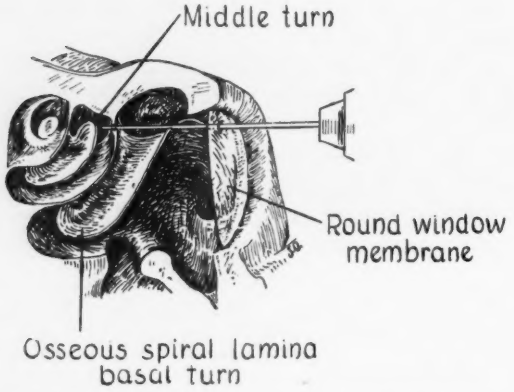

Fig. 4.—Drawing showing the method of producing cochlear lesions in this experiment. The needle is held in a pin vice and passed through the round window membrane into the osseous spiral lamina of the basal turn. In the drawing the needle is shown entering the second turn, as occurred in Cat 7 (Fig. 5).

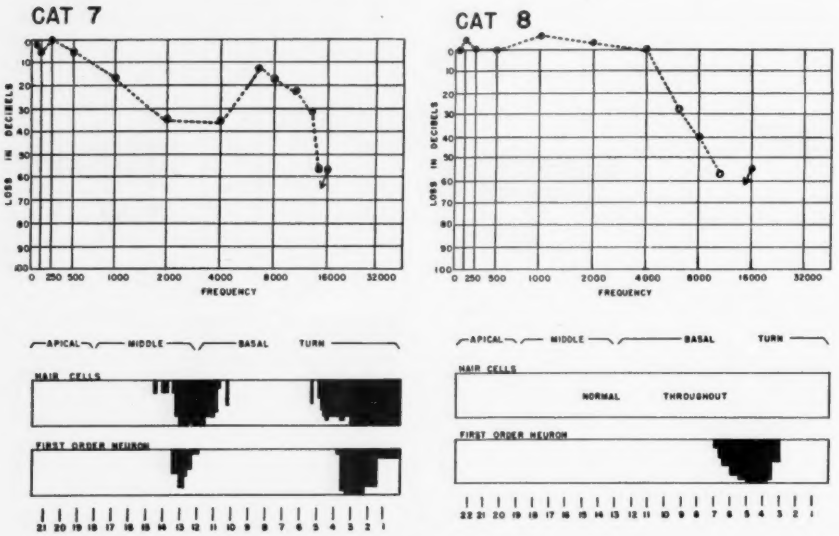

Fig. 5.—*Cat 7:* Hearing test and cochlear chart six weeks after injury was made. The needle penetrated the basal turn and entered the second turn to produce two lesions (Fig. 4). *Cat 8:* Audiogram and cochlear chart of animal sacrificed four weeks after the lesion was produced. In this ear there was damage to the spiral ganglion without a corresponding lesion of the organ of Corti.

In plotting the frequencies along the abscissa of the audiogram we have employed the "anatomical frequency scale." [8] For the guinea pig this type of scale corresponds closely to the distribution of frequencies along the cochlear partition as determined by electrical recording in animals subjected to cochlear lesions.[9] The scale is logarithmic above 900 cps and linear below. Each 100-cycle interval below 900 is equal to the distance between 900 and 1,000 on the logarithmic scale. It should be understood that the scale is hypothetical for the cat and that some error in the placement of frequencies may exist. It has been found useful, nevertheless, for correlating hearing loss with inner ear pathological findings in this type of experiment.

The cochlear charts were derived from the spiral graphic reconstructions. These linear charts are placed underneath the audiograms so that the basal end lies below 48000 cps and the apical end below 30 cps on the audiogram.

The black filling in the cochlear charts indicates the estimated percentage of hair cells and spiral ganglion cells which have degenerated. The numbers under the charts show the distance in millimeters from the basal end of the cochlea as measured along the line of union of the pillars.

We have chosen 48000 cps as the upper limit of the audible frequency scale because cortical potentials can be consistently recorded from the auditory cortex of the normal cat to tone pulses as high as 45000 to 50000 cps.[6] The lowest audible frequency for the cat is less well known but probably is less than 50 cps. Békésy has determined the lowest frequency that still has a point of maximum displacement along the cochlear duct for a number of animals, but unfortunately the cat was not included.[10] We have selected 30 cps for the cat on a purely arbitrary basis. A small error in determining this point is not serious because in this type of chart there is a crowding of frequencies at the apical end.

RESULTS

CAT 3.—A needle was introduced into the cochlear duct 2 mm. from the basal end. The animal was sacrificed 10 weeks later. The final hearing tests showed normal hearing for the frequencies tested from 62.5 to 10000 cps (Fig. 2). There was a loss of 27 db. for 12000 cps and 68 db. for 14000 cps. There was no response to 16000 cps when delivered at an intensity of 64 db. above the preoperative threshold.

Histological study revealed a loss of all the outer hair cells and 30% of the inner hair cells in a 6.8 mm. region beginning at the basal end of cochlea. Sixty per cent of the spiral ganglion cells were missing in the region between 1.5 and 2.5 mm.

As 10000 cps was the highest frequency at which hearing was good, it appears that its point of maximal stimulation must lie immediately outside the lesion or in about the 7 mm. area in this cochlea. The frequency 14000 cps represents the lowest frequency for which the animal was severely deafened, and therefore its point must be within the lesion, probably near the 6 mm. area. By the same reasoning it follows that the 16000 cps frequency is located still further basalward.

CAT 4.—In this animal the needle injured the cochlear duct and osseous spiral lamina at a point 4 mm. from the basal end. The cat was sacrificed five weeks after the operation. The

8. Davis, H.; Gernandt, B. E.; Riesco-MacClure, J. S., and Covell, W. P.: Aural Microphonics in the Cochlea of the Guinea Pig, J. Acoustical Soc. America **21**:502-510, 1949.

9. Stevens, S. S.; Davis, H., and Lurie, M. H.: The Localization of Pitch Perception on the Basilar Membrane, J. Gen. Psychol. **13**:297-315, 1935.

10. Békésy, G. V.: Über die mechanische Frequenzanalyse in der Schenecke verschiedener Tiere, Akustische Ztschr. **9**:3-11, 1944.

audiogram showed normal hearing for all frequencies tested from 125 to 4000 cps (Fig. 2). There was 32 db. loss for 8000 cps, but no response was obtained at the frequencies from 10000 to 16000 cps at the highest intensities which could be produced above the animal's preoperative threshold.

Study of the inner ear showed almost total loss of hair cells in an area extending from the basal end to the 4 mm. area and then progressive improvement to normality at the 6 mm. point.

Penetration of the osseous spiral lamina by the needle caused direct injury to the nerve fibers and resulted in a severe loss of spiral ganglion cells in the region between 3.8 and 4.5 mm.

The moderate threshold changes for the 8000 cps frequency suggest that its point of maximum sensitivity lies near the margin of the lesion or about 6 mm. from the basal end. The point for the 10000 cps frequency apparently is located within the lesion near the 5.8 mm. area in this ear.

CAT 5.—Three weeks following the operation to produce a lesion in the basal end of the cochlea the auditory thresholds were normal in the range tested (62.5 to 16000 cps). Presumably the injury was restricted to the extreme basal end of the cochlea, which serves the very high frequencies which were out of range of the testing equipment used. At a second operation (3½ weeks after the first) a large granuloma was found in the round window niche. It was removed before the lesion was made. When the animal was sacrificed six weeks after this second operation, the granuloma was found to have recurred and again filled the round window niche.[11] The final hearing tests consistently showed a loss of about 30 db. for the frequencies tested from 62.5 to 4000 cps (Fig. 3). There was a loss of 68 db. for 6000 cps, and at the frequencies 8000, 12000, and 16000 cps there was no hearing at maximum intensity.

There was a severe lesion of the sensory elements in the lower 10.3 mm. of the cochlea. There was a partial loss of spiral ganglion cells in the area between 2.8 and 5.8 mm.

As there was no inner ear lesion to explain the hearing loss for the frequencies up to 4000 cps, we assume it was a conduction loss on the basis of the pathological condition of the round window. This conduction lesion probably contributed also to the hearing losses recorded for frequencies above 4000 cps. Nevertheless, there is a 36 db. difference in threshold between 4000 cps and 6000 cps. This fact strongly suggests that in this ear the point of maximal stimulation for 4000 cps lies immediately outside the lesion in about the 10.5 to 11 mm. area and for 6000 cps near the periphery of the lesion in the 9 to 10 mm. area.

CAT 6.—This animal was sacrificed 5½ weeks after the needle was introduced into the cochlear duct at a point 5 mm. from the basal end. The final hearing tests showed the thresholds for frequencies of 62.5 to 1000 cps to be within normal limits (Fig. 3). There was a loss of 19 db. for 2000 cps, 39 db. for 4000 cps, and 91 db. for 6000 cps. At the frequencies 8000 cps and 16000 cps there was no hearing at maximum intensity.

Histological study of the inner ear revealed almost total loss of hair cells up to 9.9 mm. There was complete loss of spiral ganglion cells between 4.8 and 5 mm., with a progressive improvement to normality between 5 mm. and 8.8 mm. (Figs. 6, 7, and 8).

The 19 db. loss for the 2000 cps frequency is probably significant but cannot be explained. A loss of 39 db. for 4000 cps suggests that this frequency has its point of maximal stimulation near the periphery of the lesion, about 9.8 mm. from the basal end of the cochlear duct. There was a 91 db. loss in acuity for the 6000 cps frequency, so its point must be well within the area of the lesion, probably 8.5 to 9.5 mm. from the basal end.

11. We quote directly from our notes made at the time of autopsy: "Granuloma removed from round window niche. The round window membrane was scarred and thick. The middle ear was clean."

CAT 7.—In this animal, as in Cat 5, the first operative procedure failed to produce a hearing loss in the frequencies tested from 62.5 to 16000 cps. Three weeks later the operation was repeated. This time the needle was sharpened to a three-sided point, introduced through the round window, and used as a manual drill, so that it entered the second turn of the cochlea after penetrating the basal turn (Fig. 4). The cat was sacrificed six weeks later.

The final hearing test showed a loss of acuity in two regions of the auditory spectrum which correspond to the lesions of the two turns (Fig. 5). The thresholds for frequencies from 62.5 to 500 cps were normal. There was a loss of 18 db. for 1000 cps and losses of 35 db. for both the 2000 and the 4000 cps frequencies. The losses for the frequencies 6000, 8000, and 10000 cps were 12, 18, and 22 db., respectively. Acuity for 12000 cps was depressed by 31 db. and for 14000 cps by 57 db. At the frequency 16000 cps there was no hearing at maximum intensity.

In the spiral graphic reconstruction of the cochlea the lesions in the first and second turns were found to lie adjacent to each other. In the lesion located near the basal end there was a total loss of hair cells for a distance of 3 mm., inner hair cells only were present between 3 and 4.5 mm., and from 4.5 to 5.5 mm. the number of hair cells progressively increased to normal. The lesion in the spiral ganglion extended from the basal end for a distance of 3.8 mm. There was complete degeneration of the ganglion cells between 2.2 and 3.4 mm.

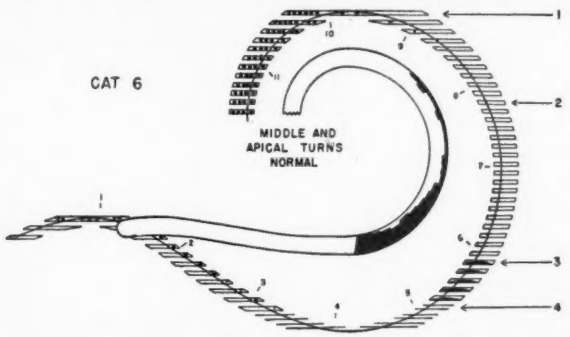

Fig. 6.—Spiral graphic reconstruction of the cochlea of Cat 6. The quadrilateral boxes represent the organs of Corti, and the dots represent hair cells. A large dot is a normal hair cell, and a small dot is an abnormal hair cell. The black mounds within the boxes represent flattened organs of Corti. The quadrilateral boxes with open ends indicate that the organ of Corti is missing. The central spiral channel represents the spiral ganglion, and the black filling indicates the estimated percentage of missing spiral ganglion cells. The numbers between this channel and the organs of Corti indicate the distance in millimeters from the basal end of the cochlea. The numbers along the right hand side of the diagram show the points from which the photomicrographs were taken, which appear in Figures 7 and 8.

The pathological state of the second lesion consisted of injury to hair cells between 10 and 14.5 mm., with a complete loss of these elements between 11.5 and 13 mm. There was a partial loss of spiral ganglion cells between 11.9 and 13.5 mm.

The 18 db. loss for 1000 cps is probably significant but at this time cannot be explained on the basis of the inner ear pathological findings. The shape of the audiogram suggests that in this ear the frequency 2000 cps has its point of maximum stimulation near the apical margin of the lesion at about 13 to 14.5 mm. and 4000 cps near the basal margin of the lesion around 10 to 11.5 mm.

The small losses for 6000, 8000, and 10000 cps are probably significant but cannot be explained. The precipitous drop of the audiogram between 10000 and 14000 cps suggests that in this ear the point for 10000 cps is near the edge of the injury at about 6.3 mm. and that the point for 14000 cps lies within the injury, about 4.8 mm. from the basal end of the cochlea.

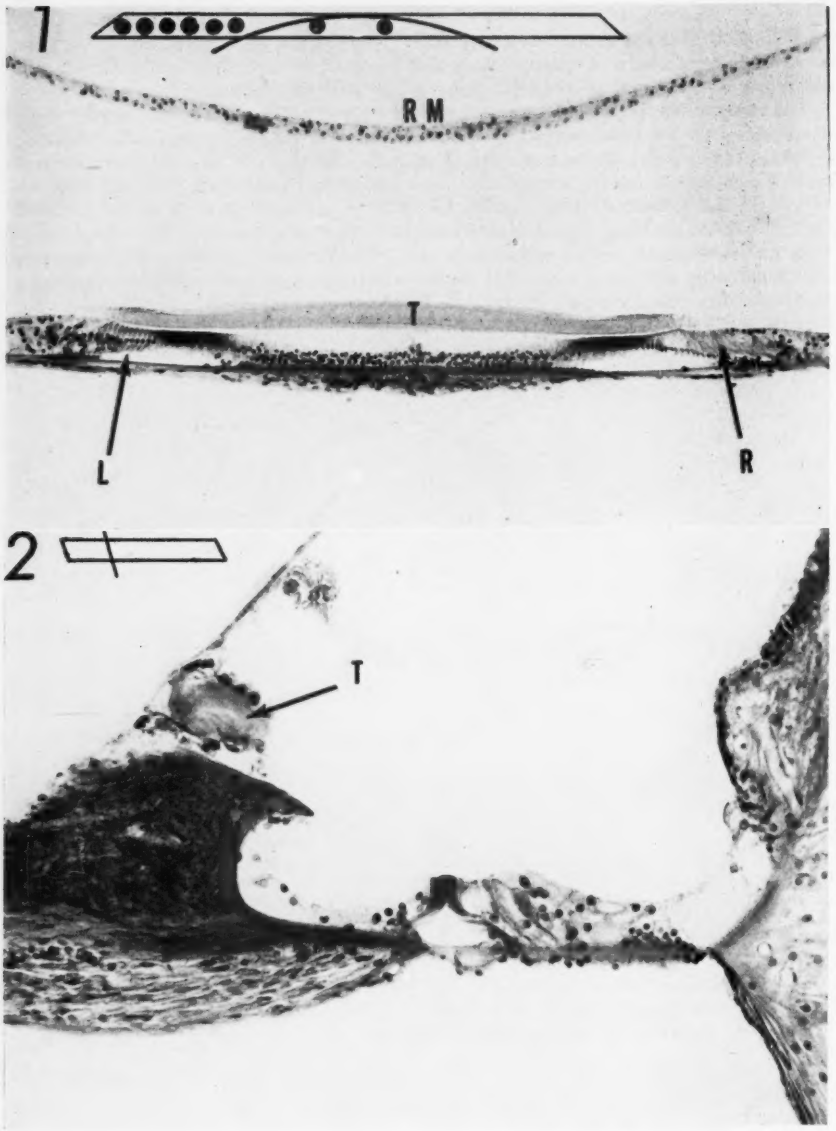

Fig. 7.—*1*, Tangential section through the upper part of the basal coil indicated as point *1* in Figure 6. This section is taken in the area of transition from normal to abnormal; note the normal outer hair cells in the organ of Corti on the left (*L*) and the absence of outer cells in the organ of Corti on the right (*R*); *RM* is Reissner's membrane, and *T* is the tectorial membrane. *2*, This section is taken from point *2* as indicated in the diagram in Figure 6. The organ of Corti is devoid of hair cells; the tectorial membrane (*T*) has been displaced and is partially surrounded by connective tissue.

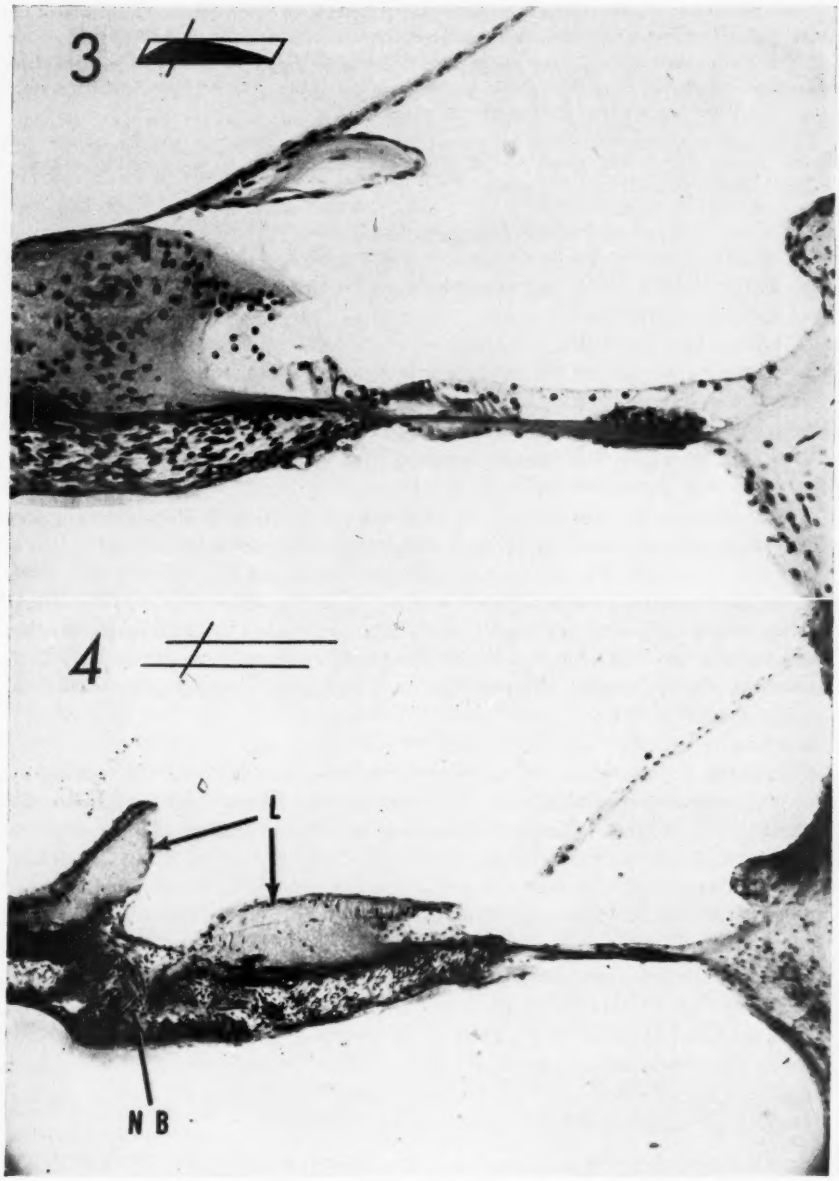

Fig. 8.—*3*, Section indicated as point 3 in the diagram of Figure 6. The organ of Corti consists of a mound of tissue of deranged architecture and devoid of hair cells; the tectorial membrane is adherent to Reissner's membrane. *4*, This photomicrograph is taken at point *4* in the diagram of Figure 6; the organ of Corti and the tectorial membrane are missing; Reissner's membrane is torn; the needle passed through the osseous spiral lamina and the limbus (*L*), separating the latter into two parts; the fracture of the osseous spiral lamina is healed by new bone (*NB*).

CAT 8.—In this ear the needle was directed into the osseous spiral lamina 4.5 mm. from the basal end. After four weeks final audiograms were made, and the animal was sacrificed.

The hearing thresholds for the range from 62.5 to 4000 cps were normal (Fig. 5). There were losses of 29 db. for 6000 cps, 40 db. for 8000 cps, and 57 db. for 10000 cps. At the frequency 16000 cps there was no hearing at maximum intensity.

Histological examination revealed normal hair cells throughout the cochlea. There was severe injury of the spiral ganglion in the area between 3 and 7 mm., with total degeneration of the ganglion between 3.8 and 5 mm.

By applying the same kind of reasoning as was used for the other animals it appears that the 6000 cps frequency lies near the edge of the lesion at about 7 mm. and that 8000 and 10000 cps are deeper within the lesion, possibly at about 6.5 and 6 mm., respectively.

It should be stated that a loss of neural elements in a particular region of the cochlea may cause defects in auditory function that differ from those which result from a loss of sensory cells in the same area. It is known, for example, that the outer hair cells are innervated by fibers from the spiral ganglion which proceed radially to the organ of Corti and then run both ways in a spiral direction for distances of several millimeters.[12]

Nevertheless, the assumptions as to frequency location in this cat correspond fairly well with the other animals. Possibly the deviation from normal is not as abrupt as in some of the animals, for example, Cats 3 and 7.

In all the animals some degree of injury to nerve fibers was created directly during the procedure of introducing the needle into the cochlea. Consequently, it is possible that the hearing losses in the other animals are reflections, in part, of the neural injury, rather than the result entirely of sensory degeneration.

COMMENT

It is valid to correlate the inner ear pathological findings with the hearing loss for each animal only if no changes are created in the sound-conducting mechanism during the experiment. Gross and histological examination of the ears revealed significant middle ear pathological conditions only in the ear of Cat 5. The audiogram of this animal was the only one to show a depression of auditory acuity for the low frequencies. It seems likely, therefore, that the audiograms of the remaining five ears reflect exclusively changes produced in the inner ear.

The needle was introduced into the inner ear of each animal so as to penetrate the osseous spiral lamina near its tympanic lip; thus, both the nerve fibers and the organ of Corti were directly injured. Fragments of the fractured osseous spiral lamina were found to be surrounded by a small amount of new bone. In several ears small displaced splinters of the bone were found lying against the wall of the scala tympani, partially embedded in loose connective tissue.

The basilar membrane was found to be intact in all ears. Reissner's membrane was torn throughout a portion of the basal coil in all ears except in Cat 8. The spiral ligament, stria vascularis, tectorial membrane, and limbus showed marked pathological changes in the region of the injury but were normal throughout the remainder of the cochlea.

12. Fernandez, C.: The Innervation of the Cochlea (Guinea Pig), Laryngoscope **61**:1152-1172, 1951.

Usually the degenerative change in the spiral ganglion was limited sharply to the region of injury, so that the transition from total degeneration to complete normality often occurred within 1 mm.

In all but one of the animals the organ of Corti, and particularly the hair cells, showed degenerative changes which were more widespread than those occurring in the spiral ganglion. The lesions of the organ of Corti and sensory cells spread for a distance of several millimeters in both directions from the point of maximum injury. The pathological states of the organ of Corti from the mildest at the periphery of the lesion to the severest in the center of the lesion can be listed as follows: (1) an organ of Corti with inner hair cells but without outer hair cells; (2) presence of an organ of Corti having supporting cells but no hair cells; (3) organ of Corti replaced by a mound of unorganized cells, and (4) total absence of the organ of Corti.

Representative photomicrographs appear in Figures 7 and 8. Near the margins of a lesion it was not unusual to find a normal number of spiral ganglion cells, although the organ of Corti was devoid of hair cells.

In one animal (Cat 8) the injury occurred to the osseous spiral lamina without involving the cochlear duct. There was severe degeneration of the spiral ganglion in the 3 to 7 mm. area, although the organ of Corti remained normal throughout the cochlea. Evidence from other experiments further supports the contention that neural degeneration can occur without a corresponding change in the sensory elements innervated.[13]

The length of the organ of Corti as measured along the junction of the pillars varied from 20 mm. for Cat 2 to 23 mm. for Cats 3 and 8. It is not definitely known whether this difference is due to actual variation in cochlear length or whether it represents errors arising in the method of graphic reconstruction. In this connection we may note that when one compares frequency localization for different cochleas, discrepancies of 1 or 2 mm. may arise in the localization of the point of maximum excitation for a particular frequency. The discrepancy is usually in the direction of placing the point of maximum excitation farther from the basal end in the longer cochleas. For example, the point of maximum excitation for the 10000 cps frequency appears to be near 5.8 mm. in Cat 4 whose cochlea measures 21 mm. and near 7 mm. in Cat 3 which has a 23 mm. cochlea.

Although an analysis of the experimental results does not allow a definite pinpointing of places of maximum stimulation for the various frequencies, it is possible to give some approximations.

From the available information it appears that 14000 cps has its maximum excitatory effect in about the 5 mm. area, 10000 cps at 6.5 mm., 6000 cps at 9 mm., 4000 cps at 10.5 mm., and 2000 cps at 13.5 mm.

The slope of the hearing curve also supplies information on the spread of the excitatory process with increase in stimulus intensity. The steeper the slope, the less is the spread. An examination of the audiogram of Cat 3, for example, suggests that for the frequency 14000 cps the excitatory process remains restricted within a 3 or 4 mm. area for intensities up to 70 db. above threshold.

Animals with low-frequency hearing losses resulting from apical lesions have audiograms with a more gradually sloping curve. The audiogram for Cat 1 shows

13. Wever, E. G., and Neff, W. D.: A Further Study of the Effects of Partial Section of the Auditory Nerve, J. Comp. & Physiol. Psychol. **40**:217-226, 1947. Schuknecht, H. F.: Unpublished data on experimental section of the cochlear nerve.

that responses were made for the lowest frequencies tested at intensities of 35 db. In this ear it appears that at an intensity of 35 db. the low frequencies are capable of exciting receptors at least 5 mm. from the point of maximum excitation. Thus, the patterns of excitation appear to be broad for low frequencies and narrow for high frequencies.

SUMMARY

For each of six cats a lesion was made in the basal coil of the cochlea by directing a small needle through the round window membrane. The effect on auditory function was determined by the conditioned-response method.

The audiograms for all animals revealed abrupt hearing losses for the high frequencies.

In five cats the inner ear pathological findings consisted of degenerative changes in the organ of Corti and spiral ganglion in a region of the basal turn. In one animal the lesion involved the spiral ganglion without injury to the organ of Corti.

The results obtained indicate that for the cat the points of maximum excitation for the frequencies 2000 to 14000 cps are arranged in an orderly manner in the area from 13.5 to 5 mm. from the basal end of the cochlea.

The experimental findings show that with increasing intensity the spread of the excitatory process is less for high frequencies than for low frequencies.

Dr. W. D. Neff of the department of experimental psychology of the University of Chicago assisted in the auditory testing of animals, and Dr. Joseph Hind and Mr. Robert Kimura helped in the technical problems. The project was aided by the suggestions and support of Dr. John R. Lindsay.

NONEXPERIMENTAL NASAL AND PARANASAL PATHOLOGY IN HEREDITARILY OBESE MICE

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OVERWEIGHT is found, according to Jolliffe,[1] to be present in 28% of the people of the United States. Data for Massachusetts gathered by Ryder and Getting [2] in a health protection clinic showed that excess weight was more frequent by far than any other pathological condition discovered in their institute.

With these facts in mind animal experimentation, long used to gain insight into the origin of this condition, was greatly promoted after the discovery was made that obesity could be produced in normal animals by bilateral lesions of the hypothalamus.

After a high incidence of overweight was observed in persons with sinus complaints (Kelemen [3]), it was thought to be of interest to examine the nasal cavities of animals with experimentally created obesity. Dr. John Brobeck, of the Department of Physiology, Yale University Medical School, collaborated by releasing heads of rats which became obese after appropriate surgical intervention. Although pathological changes were found in the nasal and paranasal cavities, similar findings were equally encountered in the numerous controls (Kelemen and Sargent [4]).

In 1950, Ingalls, Dickie, and Snell [5] described a new mutation in the V stock of the house mouse at the Roscoe B. Jackson Memorial Laboratory in Bar Harbor, Maine. These animals increase rapidly in weight until they are about four times the weight of normal animals. Young adult obese mice weigh 38 to 56 gm., whereas the weight of young adult nonobese mice is in the 16 to 26 gm. range (Mayer, Dickie, Bates, and Vitale [6]). Obesity is inherited as a simple Mendelian

This project was aided by a grant from the William F. Milton Fund.

From the Department of Otolaryngology, Harvard Medical School, and the Massachusetts Eye and Ear Infirmary.

1. Jolliffe, N.: Reduce and Stay Reduced, New York, Simon & Schuster, Inc., 1952.

2. Ryder, C. F., and Getting, V. A.: Preliminary Report on the Health Protection Clinic, New England J. Med. **243**:277-280 (Aug. 17) 1950.

3. Kelemen, G.: Sinusitis and Obesity, Laryngoscope **53**:288-304 (April) 1943.

4. Kelemen, G., and Sargent, F., Jr.: Nonexperimental Pathologic Nasal Findings in Laboratory Rats, Arch. Otolaryng. **44**:24-42 (July) 1946.

5. Ingalls, A. M.; Dickie, M. M., and Snell, G. D.: Obese, a New Mutation in the House Mouse, J. Hered. **41**:317-318 (Dec.) 1950.

6. Mayer, J.; Dickie, M. M.; Bates, M. W., and Vitale, J. J.: Free Selection of Nutrients by Hereditarily Obese Mice, Science **113**:745-746 (June 29) 1951.

recessive. Bleisch, Mayer, and Dickie [7] saw diabetes appear around the ninth week of life. The obese mice exhibit larger, more numerous islands of Langerhans; some show cutaneous atrophy and ulcers which do not heal. So far no investigations have been made regarding the other tissues of these animals.

Fig. 1.—Recess between nasoturbinal and maxilloturbinal region, filled with pus. Thin layer of exudate along epithelial surface (about 65 ×).

It seemed possible that examination of the nasal and paranasal cavities of mice from the hereditarily obese stock might furnish some information that could not be obtained from the rats with thalamic obesity mentioned above. From Dr. Meredith N. Runner of the Jackson Laboratory the heads of 10 obese mice killed

7. Bleisch, R.; Mayer, J., and Dickie, M. M.: Familial Diabetes Mellitus in Mice, Associated with Insulin Resistance, Obesity, and Hyperplasia of the Islands of Langerhans, Am. J. Path. **28**:369-385 (May-June) 1952.

by cervical dislocation were obtained preserved in a solution of formalin. Their ages varied from 53 to 85 days, and they were, therefore, in around the ninth week of life, when the manifestations of diabetes can be expected. Their weight was between 30 and 44 gm. After removal of the brain case, the brain, and the mandible, the remaining block was embedded in paraffin and sectioned in the

Fig. 2.—Lateral (ethmoid) recess, filled with pus (about 100 ×).

frontal plane. Proceeding from frontal to caudad, between 33 and 68 sections were cut and stained with hematoxylin and eosin from each specimen as a test series covering the entire length of the nasal cavities.

The findings on rat heads as reported in a previous communication (Kelemen and Sargent [4]) were considered satisfactory as controls. The olfactory organ of

the mouse is analogous to that of the rat, with reduction in size (Kolmer [8]). According to Lucas and Douglas,[9] the lateral wall of the nose of the mouse is constructed similarly to that of the rat, except that the first fold of the ethmoid concha is relatively larger; the spaces opening into the ethmoid meatuses are proportionally wider, and the gap between the opposing edges of the superior and

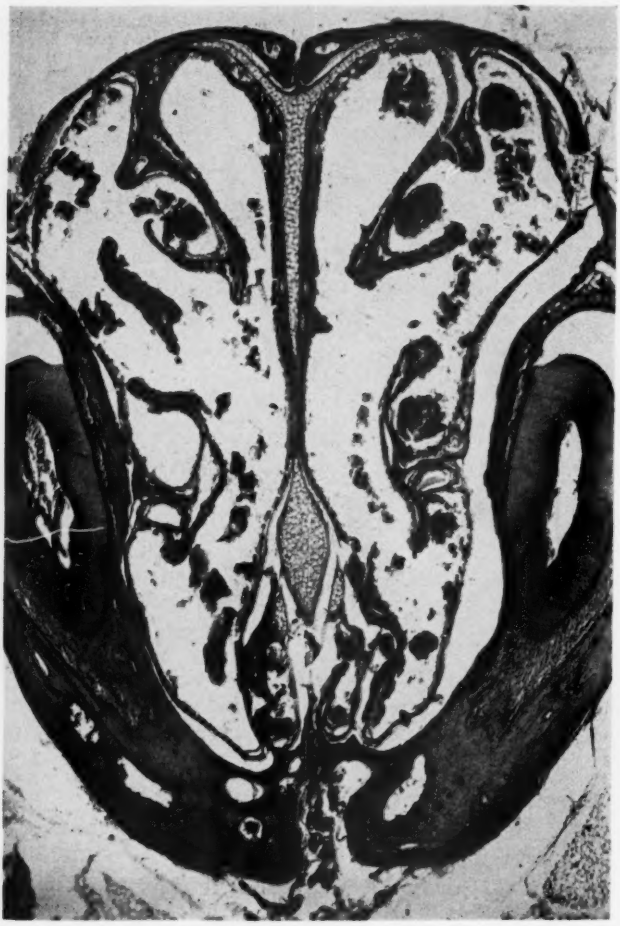

Fig. 3.—Hemorrhagic exudate in all chambers (about 40 ×).

8. Kolmer, W.: Normale Anatomie der Nase, in Jaffé, R., Editor: Anatomie und Pathologie der Spontanerkrankungen der kleinen Laboratoriumstiere: Kaninchen, Meerschweinchen, Ratte, Maus, Berlin, Springer-Verlag, 1931.

9. Lucas, A., and Douglas, L. C.: Principles Underlying Ciliary Activity in the Respiratory Tract: II. A Comparison of Nasal Clearance in Man, Monkey, and Other Mammals, Arch. Otolaryng. **20**:518-541 (Oct.) 1934.

inferior conchae is greater, while the pattern of ciliary currents is practically a duplicate of that of the rat; the ostium of the maxillary sinus is located closer to the middle meatus than in the rat, and as a result, a larger portion of the mucus from the sinus passes into this meatus or along the edges of the conchae.

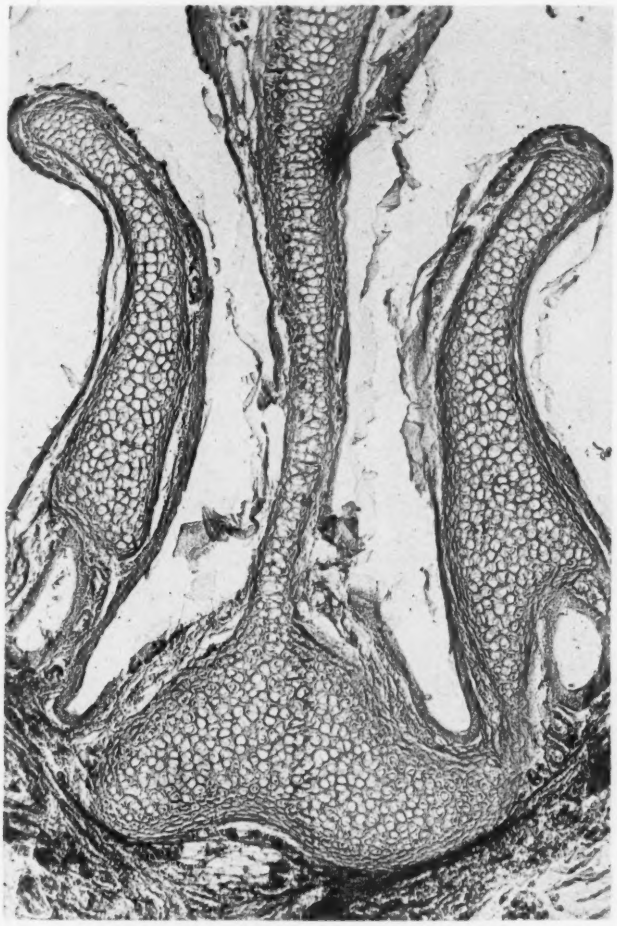

Fig. 4.—Anterior part of nasal chamber, filled with foreign (plant) material (about 100 ×).

The septal window, as described for the first time in two previous papers (Kelemen and Sargent[4] and Kelemen[10]), was found in the same location and showed the same proportions as in the rat.

Mucus on the epithelial surface and fibrin lining the latter or found in the chambers, if not in excessive quantity, were considered as normal. Comparative

10. Kelemen, G.: The Junction of the Nasal Cavity and the Pharyngeal Tube in the Rat, Arch. Otolaryng. **45**:159-168 (Feb.) 1947.

and human nasal pathology shows the readiness of the epithelial lining to separate from its basis or to undergo destruction on fairly slight provocation by irritative processes. However, as separation of or damage to the very delicate epithelium is one of the seemingly often inevitable artifacts in certain specimens, the precaution was used here not to tabulate similar findings as pathological changes. With only

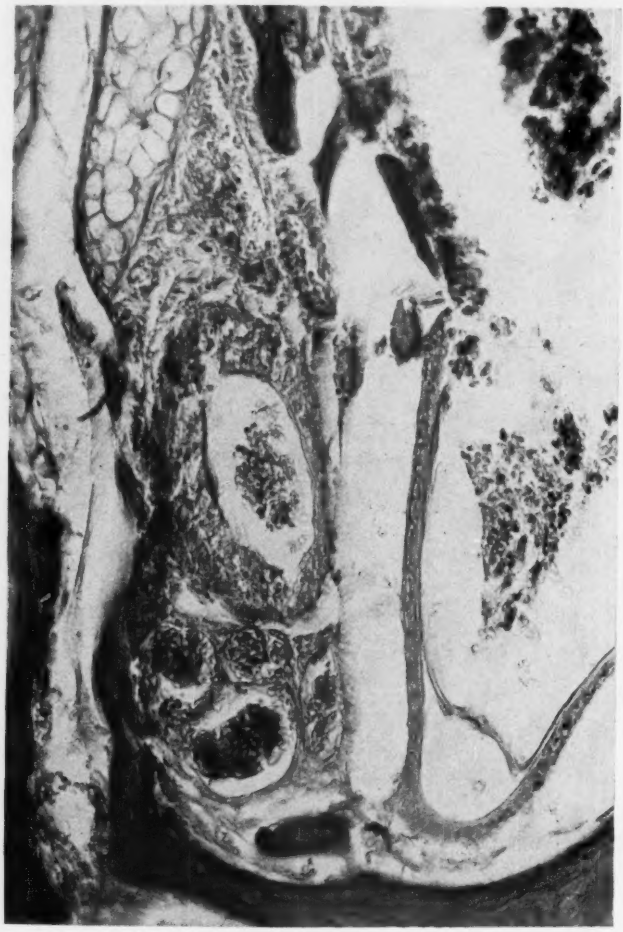

Fig. 5.—Organ of Jacobson. Pus in lumen of the duct, engorgement in the vascular plexus (about 270 ×).

three of the animals free of such the following abnormal findings were noted in the nasal cavity and the maxillary sinus, the latter being the only paranasal sinus present in mice.

In the main cavity

Mucus, in excessive amount, two cases

Fibrin, in excessive amount, two cases

Suppuration
- In the lateral ethmoidal recess, one case
- In the common meatus, one case
- In the duct of Jacobson, one case

Hemorrhagic exudate in all chambers, one case

Engorgement in the vascular plexus of the organ of Jacobson, two cases

Foreign (plant) material, one case

Fig. 6.—Organ of Jacobson. Engorgement in the vascular plexus. Duct free (about 270 ×).

In the maxillary sinus
- Excess interstitial fluid (edema) between the mucous membrane and the bony wall, one case
- Suppuration, two cases

Although the number of the examined heads (10) was too small to compute proportions of incidence, the findings give an illuminating cross section of the situation. In 7 out of 10 considerable pathological changes existed in the nasal cavity

and the maxillary sinuses. The incidence of similar conditions in apparently normal rats was found to be 60% (Kelemen and Sargent [4]). This is very nearly the same proportion for the two groups.

On the basis of these findings, the same warning as in the case of rats must be given should the olfactory organ of these obese mice be used in experimental work. The results of intranasal experimentation with drugs introduced into the cavity in

Fig. 7.—Hemorrhagic exudate in the maxillary sinus (about 270 ×).

pharmacology or of procedures on the basis of olfactory cues in comparative psychology are to be considered valid only when they are followed by careful histological analysis, including control groups, as to preexisting nasal and sinusal pathological conditions. In the consideration of experiments working with olfactory discrimination, the changes found here within the organ of Jacobson acquire a special significance.

In the mouse, as in the rat and in the guinea pig, one side of the nose cannot be used as a control against the medicated, other side because the two halves communicate through a physiological window near the base of the septum nasi.

SUMMARY

Histological examination of serially sectioned nasal cavities from the hereditarily obese V stock of the house mouse developed in the Roscoe B. Jackson Laboratory at Bar Harbor, Maine, showed considerable nonexperimental pathological changes in 7 individuals out of 10. Consequently, it is suggested that results of pharmacological or psychological experiments using the olfactory organ of similar animals be considered valid only when subsequent histological analysis does not uncover some preexisting pathological condition.

CLINICAL ASPECTS OF TUMORS OF MAXILLARY SINUS

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TUMORS of the maxillary sinus, excluding the various cysts found in this region, are relatively uncommon in the experience of most rhinologists. Probably less than 2% of all malignant tumors of the upper respiratory and alimentary tracts involve this structure. Many benign tumors are even more rarely observed. Sjöberg, for example, up to 1934 was able to collect 343 cases of osteoma of the sinuses, mostly involving the frontal group, but Rawlins [1] reviewing the world literature up to 1940 found only 32 reported cases of osteoma of the maxillary sinus. Reports of chondroma, myxoma, and fibroma are correspondingly infrequent. These various tumors not only exhibit clinical and pathologic characteristics of considerable interest but may also present complicated problems in diagnosis and management.

PATHOLOGIC BACKGROUND

The embryologic development of the upper teeth and maxilla introduces both ectodermic and mesodermic elements in complex formations and favors the occurrence of cell rests of both varieties. These, together with the mucous membrane lining the antrum, form the source of the widely varied group of benign and malignant tumors found in this area. Benign tumors include osteoma, fibrous osteoma, osteochondroma, myxoma, and an interesting group of tumors of dental origin, including adamantinoma (ameloblastoma) and odontoma of simple and mixed varieties. Other tumors occasionally reported include lipoma, psammoma, meningioma, rhabdomyoma, neurofibroma, and teratoma. The antrum may occasionally be partially or completely obliterated by the sclerotic formations of that curious development anomaly fibrous dysplasia of bone. The upper respiratory tract is the site of predilection for the extramedullary forms of plasma cell tumors, and involvement of the maxillary sinus by plasmocytoma is occasionally observed. Mixed tumors of aberrant salivary gland origin have also been reported. The majority of malignant tumors of the antrum are of the moderately differentiated epidermoid or squamous-cell variety, although adenocarcinoma and transitional-cell and basal-cell carcinoma are occasionally encountered. Primary sarcoma of the maxillary sinus is rare. In the series of 220 cases reported by Wille [2] only 6.8%

Read at the annual meeting of the International College of Surgeons, Chicago, Sept. 4, 1952.

From the Department of Surgery, Division of Otolaryngology, University of Rochester School of Medicine and Dentistry.

1. Rawlins, A. G.: Osteoma of the Maxillary Sinus, Arch. Otolaryng. **32**:499, 1940.

2. Wille, C.: Malign Tumours in the Nose and Its Accessory Sinuses, Acta oto-laryng., Supp. 65, p. 5, 1947.

were of sarcoma of various types, and Windeyer [3] reporting on a series of 153 consecutive cases of malignant tumor found only 12 instances of sarcoma. It is of interest to observe that the maxillary sinus is involved by primary cancer five times as frequently as all the other sinuses combined. The antrum is also commonly invaded by tumors originating in adjacent areas, notably the nose, nasopharynx, and upper alveolar process. Metastatic invasion has been occasionally reported from malignant tumors of the thyroid, breast, gastrointestinal tract, kidney, prostate, and long bones.

Certain of the tumors mentioned exhibit pathologic characteristics which are of sufficient clinical importance to merit a brief discussion. Adamantinoma [4] (ameloblastoma) may be described as a central tumor, slow and intermittent in its growth, anatomically benign, but clinically persistent. Both solid and cystic varieties occur, the majority (80%) being cystic. Although this tumor arises from the enamel organ, it never contains enamel. Adamantinoma is properly classified as benign, but its relentless and persistent recurrence if incompletely removed as the result of microscopic finger-like extensions into the surrounding bone indicates that surgical removal must be thorough and radical if recurrence is to be avoided. Simple curettage has no place in the treatment of this tumor. There is, furthermore, a proved incidence (4.5%) of malignant degeneration with true regional and distant metastases.

Chondroma, which actually occurs more commonly in the nasal septum than in the sinuses, demonstrates similar tendencies to recurrence if incompletely removed, as well as true malignant changes. Osteoma, whether of the eburnated, spongy, or mixed variety, should be regarded as a neoplasm of slow but unlimited growth which must be removed together with its base for successful outcome. The usual attachment of this tumor is by a cancellous bony pedicle, but in a small percentage a broad sessile base is present. Detachment of the tumor occasionally occurs (so-called dead osteoma), but in many of these instances, according to Simpson,[5] nutrition is actually supplied by small connective tissue roots or minute bony pedicles not detected at operation.

Extramedullary plasma cell tumors most commonly occur in the upper air passages. It is of interest to note that Hellwig's [6] review of 127 cases indicates the maxillary antrum to be the sinus most frequently involved. Hellwig found these tumors, in spite of a typical cell structure, to range from localized and benign forms to highly malignant varieties; furthermore, the clinically noncancerous phase may persist for several years and then without change in histologic character, generalized malignant lesions may develop and rapidly progress to a fatal outcome. Figi, Broders, and Havens [7] believe that these extramedullary tumors are truly malignant

3. Windeyer, B. W.: Malignant Tumors of the Upper Jaw, Brit. J. Radiol. **16**:362, 1943.

4. Heatly, C. A.: Adamantinoma of the Maxillary Sinus with a Report of 2 Cases, Ann. Otol. Rhin. & Laryng. **55**:322, 1946.

5. Simpson, W. L., and Williams, R. I.: Osteoma of the Nose and Accessory Sinuses with Report of Cases, Ann. Otol. Rhin. & Laryng. **49**:949, 1940.

6. Hellwig, C. A.: Plasma Cell Tumors as Observed in Various Locations, Arch. Path. **36**:95, 1943.

7. Figi, F. A.; Broders, A. C., and Havens, F. Z.: Plasma Cell Tumors of the Upper Part of the Respiratory Tract, Ann. Otol. Rhin. & Laryng. **54**:283, 1945.

lesions and should be classified and treated as such. In their series of 11 cases six of the lesions involved the maxillary sinus, and all of these presented the appearance and characteristics of malignant tumors. Hellwig found prognosis to be unfavorable in those cases presenting local evidence of invasion of bony structures. There seems to be no question that the inherent uncertainty of the prognosis in these cases necessitates radical treatment if favorable results are to be expected.

So-called cylindroma has been occasionally reported in the maxillary sinus, and Beck and Guttman,[8] in 1936, were able to collect 23 cases from the literature, including two cases of their own. This is actually a slow-growing locally malignant tumor exhibiting marked tendencies to recur and should properly be classified as adenocystic carcinoma. The tendency of mixed tumors to recur if incompletely removed is also well recognized, as well as the possibilities of their malignant transformation. They may occasionally occur primarily in the antrum but more commonly invade the sinus from the adjacent palate.

Certain pathologic features of malignant antral tumors also deserve mention. Most of these tumors are far advanced before clinical evidence of their presence is discovered. For this reason attempts to classify them from a prognostic standpoint according to their point of origin or location within the antrum are seldom practical. In general, however, tumors originating along the floor and in the lower portion of the antrum are more favorable for treatment and offer a better prognosis than those arising in the superior part. Malignant antral tumors are generally considered to metastasize relatively late. It may be recalled that lymphatic pathways lead anteriorly into the submaxillary and carotid bulb nodes, but the principle extension is posteriorly by way of the choanal network to the retropharyngeal and deep jugular nodes. Metastases actually occur far more frequently than is generally believed. In Watson's [9] series of 127 cases 29% presented metastases. Thirty-eight per cent of Wille's series (220 cases), 38.6% of Windeyer's series (153 cases), 61% of Taylor and Nathanson's series (28 cases), and over 30% of Cave's series presented metastases. It is evident that the percentage is sufficiently high to warrant careful examination for this complication.

CLINICAL OBSERVATIONS

The majority of tumors of the maxillary sinus develop slowly and often reach considerable size before causing well-defined symptoms. This statement applies to benign, as well as many malignant, neoplasms. The symptoms encountered in the malignant variety in their usual order of frequency include pain, swelling over the sinus (facial, alveolar, or orbital), nasal obstruction, nasal discharge, recurring epistaxis, and lacrimation. Pain in the upper teeth or the face, as well as facial paresthesia, may be a relatively early and puzzling complaint, but the others are actually evidence of advanced growth. Pain, however, may also occur in benign expanding tumors, and many of the other symptoms so characteristic of a malignant growth may also be closely simulated. The frequent occurrence of superimposed secondary infection may further obscure the diagnosis. While the clinical appearance

8. Beck, J. C., and Guttman, M. R.: Basaloma or So-Called Cylindroma of Air Passages, Ann. Otol. Rhin. & Laryng. **45**:618, 1936.

9. Watson, W. W.: Cancer of the Paranasal Sinuses, Laryngoscope **52**:22, 1942.

of a malignant tumor in this area, therefore, is often characteristic, neither the history nor the clinical examination can be expected to give the final diagnosis.

Roentgenographic studies of the sinus are of great value in diagnosis, as well as in treatment. In certain tumors, such as dentigerous cysts, adamantinoma, and osteoma, the characteristic roentgenographic findings may prove actually diagnostic. Evidence of destruction of one or more of the bony walls of the antrum is commonly found in the late stages of antral malignant tumors. Watson in a series of 112 cases found roentgenographic evidence of bone necrosis in 83%. It is important to emphasize, however, that similar changes may be found as the result of pressure erosion by expanding benign tumors, so that complete reliance cannot be placed on this finding.

Biopsy, therefore, becomes the final arbiter and the only accurate basis for diagnosis and treatment. It should be done, if possible, in every case. The common tendency of malignant tumors to invade the nasal passages facilitates this procedure, although repeated biopsies may be necessary because of superficial necrosis before representative tissue is secured. In certain instances of antral tumors, however, ready access is not available, and aspiration biopsy or surgical exposure of the tumor may be necessary. Exploration is always justified and indicated in doubtful cases. Biopsy not only permits accurate diagnosis but in malignant tumors assists in the planning of treatment by giving indication of the grade of malignancy. It is of interest to note that in Watson's series 60% of the cases of epidermoid carcinoma were classified as Grade 2.

TREATMENT

Surgical access to tumors of the maxillary sinus is provided either by the transantral approach through the buccoalveolar sulcus, combined, when necessary, with resection of the adjacent hard palate, or by an external approach employing the usual or the modified forms of lateral rhinotomy.[10] The majority of benign tumors can be successfully removed by the peroral route, except the large tumors which require external management. The importance of creating a permanent infralabial opening in cases of benign tumors with recurrent tendencies, notably adamantinoma, must be strongly emphasized. This can be secured by providing an appropriate prosthetic appliance for daily use in the fenestra. The treatment of malignant neoplasms must be individualized. The age and condition of the patient, the type and gradation of the tumor, its size and location as determined by careful roentgenograms, as well as clinical and roentgenographic studies for possible unfavorable local extensions or metastases, are all of fundamental importance. Although irradiation alone has provided survivals of five years or more, especially in the lymphosarcoma group, the results have, for the most part, been disappointing, and the ensuing complications have ultimately required surgery for drainage or removal of sequestra. Nevertheless, it is generally agreed that a small group of highly radiosensitive antral tumors, notably lymphosarcoma and transitional-cell carcinoma, are best treated by primary irradiation. Patients in unfavorable general condition and those showing widespread

10. Heatly, C. A.: Expanding Tumors of the Maxillary Sinus: Value of the Lateral Rhinotomy, New York J. Med. **51**:2640, 1951. Schuknecht, H. F.: Surgical Management of Carcinoma of the Paranasal Sinuses, Laryngoscope **61**:874, 1951.

local extensions or metastases should also be treated by primary irradiation. It is our feeling, however, that the salvage rate of antral malignant tumors will be increased by more frequent resort to radical surgical measures supplemented by the more accurate application of radium and x-ray therapy made possible by this exposure. It is, of course, true that by the time the majority of these tumors are discovered, infiltration beyond the confines of the maxilla has commonly occurred, so that a complete block resection is seldom possible. Surgery, nevertheless, accomplishes several desirable purposes: (1) It removes the bulk of the tumor; (2) it permits a more accurate appraisal of the boundaries of the neoplasm, as well as a careful search for unfavorable regional extensions; (3) in this way it makes possible the accurate use of intracavitary radium therapy or deep x-ray therapy so as to deliver the full cancerocidal dose to the areas where it is most needed, and, finally, (4) it guarantees adequate drainage for the infection and sequestration which complicate this situation. Radical surgical procedures have been greatly aided and their scope widened to include even elderly patients by the recent advances in anesthesia, especially the careful use of intravenous thiopental (pentothal®) sodium and curare, as well as the control of complicating infections made possible by the antibiotics. Surgical diathermy has also proved to be an indispensable adjunct, as has been stressed by New [11] and others. Surprisingly little reaction follows even extensive coagulation, and available evidence indicates that low-grade malignant tumors can frequently be destroyed completely by this medium.

Although combined surgery and radiotherapy now constitute the accepted treatment in antral malignant tumors, opinions concerning the advantages of employing irradiation before or after surgery vary considerably. Windeyer, Robinson,[12] and Capps and Williams [13] all urge the preliminary use of irradiation. They point out that the size and vascularity of the growth are reduced to more manageable proportions, the tendency to spread is lessened, and the effectiveness of the rays is increased by virtue of the undisturbed blood supply. Others, including New, Collins and Poole,[14] Wille, Schall, and Schuknecht, favor initial surgery followed by irradiation. This decision should properly be an individual one, and in many instances some form of irradiation both before and after surgery may be employed to best advantage.

The management of metastatic cervical glands in these cases presents a problem of great difficulty. As has been already pointed out, metastases in antral malignant tumors occur relatively late in the disease but are recorded in fully 30% of most reported series. Ogura's [15] studies indicate that in advanced malignant tumors of the larynx microscopically demonstrable metastatic carcinoma occurs in a high percentage (38%) of cervical nodes not clinically suspicious. The actual incidence in maxillary sinus cancer, therefore, must be similarly greater. The principal

11. New, G. B.: Malignant Diseases of the Paranasal Sinuses, Am. J. Surg. **42**:170, 1938.

12. Robinson, G. A.: Diagnosis, Treatment, and End Results in Malignant Tumors of the Nasal Sinuses, New York J. Med. **44**:713, 1944.

13. Capps, F. W. C., and Williams, I. G.: Discussion on Malignant Diseases of the Nasal Cavity and Sinuses, Proc. Roy. Soc. Med. **43**:665, 1950.

14. Collins, V. P., and Poole, J. L.: Treatment of Antral Cancer by Combined Surgery and Radium Therapy, Radiology **55**:41, 1950.

15. Ogura, J. H., and Bello, J. A.: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx, Laryngoscope **62**:1, 1952.

lymphatic pathways from the antrum lead posteriorly by way of the choanal network to the inaccessible retropharyngeal glands and thence to the deep jugular nodes. This situation seriously limits the usefulness of block dissection in these cases and accounts for the general reliance on irradiation, despite its well-recognized failures, except in occasional limited instances.

SUMMARY AND CONCLUSIONS

Tumors of the maxillary sinus are infrequent and widely varied. They include not only frankly benign and malignant neoplasms but also an intermediate group exhibiting strong tendencies to local recurrence. In this region, as in others, progress in surgery, as well as radiotherapy, depends in part on a sounder understanding of the natural history of these tumors, including their invading and metastasizing tendencies. The wider range of operability created by the antibiotics and the newer techniques in anesthesia, as well as improvements in irradiation therapy, should stimulate a renewed interest in the management of these tumors.

PRIMARY TUMORS OF THE FRONTAL BONE

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WHILE much work has been devoted to tumors of the ethmoidomaxillary area, primary tumors of the frontal bone, including the frontal sinus, have attracted the attention of the investigators to a less degree. The reason is that tumors of the frontal bone, as a whole, particularly those originating in the frontal sinus, do not occur as frequently as tumors of the ethmoidomaxillary area. Grégoire and Martin [1] collected, in 1948, from the literature 31 cases of primary carcinoma and 25 cases of sarcoma of the frontal sinuses. The question arises as to the cause of the difference in the frequency. This question, which is related to the fundamental question of the origin of the tumors as a whole, cannot be answered. However, a few factors can be determined to explain the difference. In the texbooks great emphasis is laid upon the exposure of the superior maxilla to various traumatisms due to the act of chewing, respiration, and others. The importance of these factors is evident. Yet, I wish to call attention to two other factors which apparently are not fully appreciated. In a certain number of normals one or both of the frontal sinuses does not develop at all. The ethmoids and the maxillary sinuses develop always in normals. For this reason the number of tumors which originate in the mucosa of the sinuses must be smaller in the frontal area. Furthermore, there is a wide "party wall" between ethmoid and maxillary sinuses on one hand and the nasal passages on the other hand, and this "party wall" is very thin. In contrast. there is only a small "party wall" between frontal sinus and nasal passages, and this "party wall," viz., the nasal spine of the frontal bone, is very thick. For this reason tumors which originate in the ethmoidomaxillary area will grow rapidly into the nasal passages, where they can be discovered without difficulty. In contrast, primary tumors of the frontal bone and the frontal sinus have a latent course for a comparatively long period until they grow into the orbit or beneath the skin of the forehead or into the skull or into the ethmoid and from the ethmoid into the nasal passages. For this reason in primary tumors of the frontal sinus no tumor or only a small amount of tumor tissue can be detected in the nasal passages for a long period. The cases of Geschelin,[2] Lachman,[3] Breeding,[4] Heindl,[5] Wilmot,[6]

Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions reached by the author are a result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Grégoire, A., and Martin, P.: Deux cas d'épithélioma primitif du sinus frontal, Oto-rhino-laryng. internat. **32**:45, 1948.

2. Geschelin, A. J.: Zur Klinik der bösartigen Neubildungen in den Nebenhöhlen der Nase: Primäres Carcinom der Stirnhöhle, Acta oto-laryng. **21**:351, 1939.

and others, as well as the case of osteogenic sarcoma reported in this paper, prove this fact. This situation is, of course, different in secondary tumors of the frontal sinus, viz., in tumors which originate in the ethmoid or in the frontal process of the maxilla and extend into the frontal sinus.

In summary, the comparative rarity of tumors of the frontal bone, and particularly of the frontal sinus, is, among other causes, due to the facts that not all human beings have a frontal sinus and that the diagnosis of this type of tumor is not infrequently difficult. For this reason two cases of tumor of the frontal bone are presented which are unusual: hemangioma and osteogenic sarcoma of the frontal bone. In addition, a few other diseases of the frontal bone are briefly discussed in order to demonstrate the difficulty of the diagnosis.

REPORT OF CASES

CASE 1.—F. M., a white woman aged 38, suffered from headache for many years, which gradually became worse. In the last week before admission she could not move her head. Headache was not localized. Occasionally she had spells of whirling dizziness. For many years she suffered from multiple subcutaneous hard "fibromas" in the left breast and in the vulva. The "fibromas" of the breast became very sore during menstruation. Three years prior to admission she noticed a hard tumor above the left eye approximately in the center of the left eyebrow. The tumor was tender and caused neuralgiform pain.

On Oct. 7, 1942, there was hyperplastic rhinitis. Approximately in the center of the left supraorbital arch there was a tumor, the size of a cherry, covered by normal skin, not movable, and tender. X-ray examination revealed normal sinuses. In the temporal area on both sides there was an increase of the number of diploic veins. Above the center of the left orbital arch there was an area of rarefaction, the size and the form of a dime, which had rather distinct margins. In this area the bone had a mottled appearance and was in connection with the venous plexus in the temporal area. The connection was accomplished by two veins. The superior margin of the orbit seemed to be depressed in this area, which corresponded to the area of the tumor visible on the patient's forehead. In the lateral exposure there was a small tumor above the frontal sinus bulging toward the skin. The tumor seemed to originate from the external lamina of the frontal bone. The tumor was radiopaque and had a homogeneous structure. The diploë and internal lamina were intact (Fig. 1).

On Oct. 26, 1942, an incision was made below the left eyebrow. The periosteum was exposed and was incised vertically. A bony tumor, the size of a cherry, was exposed. It had the appearance of a bone cyst. It was covered by a thin shell of bone which had a bluish color in contrast to the yellow color of the surrounding bone. The surrounding compact bone of the external lamina formed a calyx-like structure which contained the bulging tumor. A gutter was made around the tumor in the normal diploë, and the tumor was removed. The dura was not exposed. Uneventful recovery ensued. Subsequent to the operation the neuralgiform pain decreased.

Microscopic Examination.—The tumor was covered by firm periosteum and consisted of empty capillaries and cavernous spaces, the walls of which were made up by flat endothelium. Between the blood spaces there were bony spicules which consisted of woven or lamellar bone. On several places the lamellar bone, infrequently the woven bone, was in direct contact with the endothelium of the blood spaces; on other places there was a loose connective tissue, rich in

3. Lachman, J.: Reticulum Cell Sarcoma: Reticulum Cell Sarcoma of the Frontal Sinus, Acta med. orient. **5**:50, 1946.

4. Breeding, E. G.: Malignancy of the Nasal Accessory Sinuses with Report of 2 Cases of Primary Carcinoma of Frontal Sinuses, Ann. Otol. Rhin. & Laryng. **49**:141, 1940.

5. Heindl, A.: Über die Geschwülste der Stirnhöhle, Monatsschr. Ohrenh. **64**:128, 1930.

6. Wilmot, T. J.: Primary Malignant Disease of the Frontal Sinus, J. Laryng. and Otol. **66**:283, 1952.

nuclei, between the blood spaces and the bone spicules, and there were rows of osteoblasts which laid down osteoid on the bone spicules. There were no osteoclasts, but there were Howship's lacunae. On some places the loose connective tissue was very much developed, giving rise to a metaplastic ossification. The cavernous spaces might reach the periosteum. There was no bone marrow. The external lamina of the frontal bone, surrounding the tumor, was normal (Fig. 2).

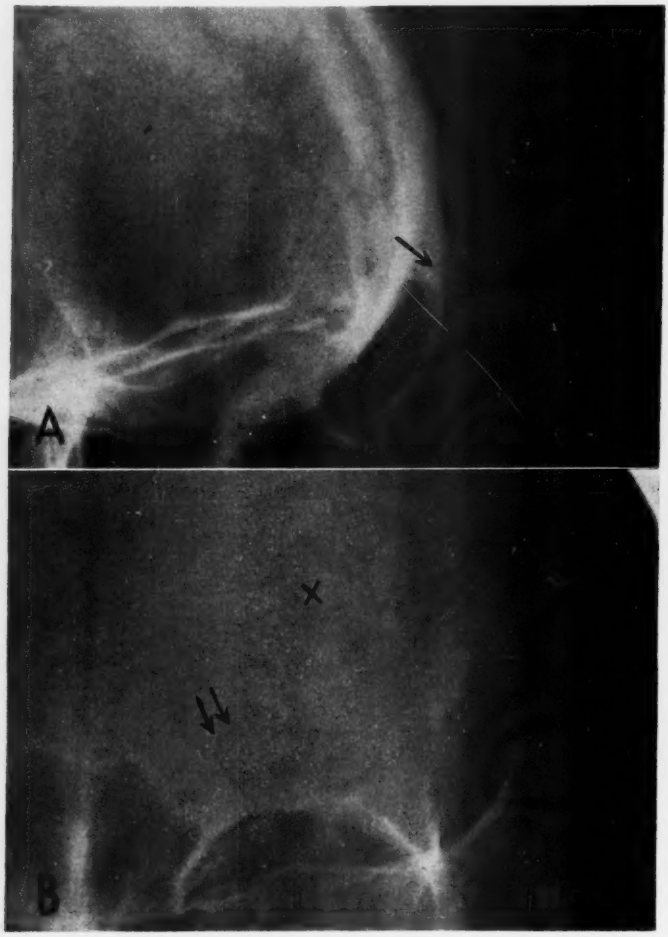

Fig. 1.—*A*, the arrow indicates the small subperiosteal tumor above the frontal sinus. *B*, *x* indicates the plexus of the diploic veins in the temporal area; the two arrows indicate the hemangioma of the bone. Note the two diploic veins which connect the hemangioma with *x*.

This was a very small hemangioma of the frontal bone close to the frontal sinus. These tumors are rare. Recently Kelemen and his co-workers [7] reviewed the respective bibliography and reported four new cases. In all these cases the tumor

7. Holmes, E. M.; Sweet, W. H., and Kelemen, G.: Hemangiomas of the Frontal Bone: Report of 3 Cases, Ann. Otol. Rhin. & Laryng. **61**:45, 1952. Kelemen, G., and Holmes, E. M.: Cavernous Hæmangioma of the Frontal Bone, J. Laryng. and Otol. **62**:557, 1948.

was larger than in the presented case and was situated in the area of the tuber frontale. In the presented case the tumor developed in the area of the orbital arch.

Microscopically, the tumor was partly a capillary, partly a cavernous, angioma. The tumor did not take its origin from the meninges or from the scalp; the x-ray film proved, rather, a connection of the tumor with diploic veins. There was in the temporal area on both sides an increase in the number of diploic veins forming a structure similar to a "parietal spider." This is not a common finding. However, since there is, according to Wischnewski [8] and Schmidt,[9] a great variability as far as the development and course of the diploic veins is concerned, this finding should not be considered pathological but the result of an abnormal anlage of the diploic veins.

Fig. 2.—Hemangioma of the frontal bone; *a* indicates the outer periosteum.

In the presented case two veins emerged from the "spider" on the left side, took a convergent course toward the frontal sinus, and ran apparently toward a round area of rarefaction which had rather distinct margins and an indistinct honeycomb structure. The area corresponded to the tumor on the patient's forehead. These findings indicate (1) that the angioma apparently took its origin from the diploic veins and (2) that the tumor was probably not a true neoplasm but a hamartomatous angioma, viz., a congenital anomaly of the diploic veins which in the course of growing developed to a tumor.

The tumor grew toward the external lamina of the frontal bone, which became expanded, very thin, and finally dehiscent so that the tumor was covered on several places by periosteum only. Microscopically, the compact bone of the external

8. Wischnewski, A.: Die Venæ diplocicæ der Schädelknochen, Ztschr. Anat. **77**:381, 1925.

9. Schmidt, H.: Osteomyelitis der platten Schädelknochen, insbesondere des Stirnbeines, Arch. Ohren-Nasen-u. Kehlkopfh. **143**:115, 1937.

lamina had been replaced by spicules of bone which were scattered between the blood spaces and separated from the latter by loose connective tissue or by the endothelium of the blood vessels. Since some of these spicules consisted of woven bone which does not occur in the mature frontal bone, it is evident that the spicules were newly formed. In fact, there was much osteogenesis, the new bone being formed by osteoblasts or by metaplastic ossification of connective tissue. There were no osteoclasts, but there were Howship's lacunae, indicating a slight degree of osteoclasis. There was no periosteal reaction, and for this reason there was no "sunray" pattern on the x-ray plate; viz., there were no radiating spicules of bone overlying the tumor, which are supposed to be significant of bone angiomas of larger size.

In the presented case the tumor was removed together with the adjacent diploë and lamina externa, but the dura was not exposed. Kelemen advises the removal of the lamina externa and diploë plus the lamina interna. In the presented case there was no recurrence of the tumor for a period of 10 years, although the internal lamina was not removed.

The tumors of the breast and the vulva were not examined. The patient's husband, a dentist, said that they had been found to be "fibromas."

In the presented case the correct diagnosis was not made prior to the operation because the x-ray films were correctly interpreted after the operation. Prior to surgery other diseases of the frontal bone were considered, namely, syphilitic periostitis and ostitis and nonspecific periostitis of the frontal bone. In both diseases the x-ray findings may resemble those obtained in the presented case. The following cases are briefly reported to prove this fact.

K. R., a Negro woman of 52 years, noticed a swelling over the left eyebrow six years prior to admission. After about 1½ years the swelling disappeared, leaving behind a scar and an indentation in the involved area. In October, 1947, a "cold" developed. From the onset of the cold there was severe pain in the area of the left frontal sinus.

On Jan. 16, 1948, there was a swelling of about 3 in. (7.6 cm.) in diameter protruding slightly in the central region of the forehead. Slightly to the left was a depression in the skull to which the overlying skin was not attached. There was a slight pulsation in the center of the depression. There was no tuberculosis. Wassermann's test four times gave positive results in the blood. X-ray showed absence of both frontal sinuses. The space of the left frontal sinus was taken by an oval area of rarefaction which had an indistinct honeycomb structure. Above this area there was another, larger area of similar structure which extended beyond the midline to the right side. There were calcifications of the falx (Fig. 3). On Jan. 17, 1948, a biopsy was done. Periosteum was thickened and stripped easily from the bone. A large defect was exposed in the frontal area. This was filled with soft tissue in which newly formed bone could be noticed. The microscopic examination revealed a large central mass of necrotic tissue in which cell outlines and structures were still evident. The blood vessels had thickened walls, and some appeared still functional. Fragments about the margins of this necrotic area were fibrous and lymphocytic, and foci of epithelioid cells with giant cells of the Langhans type were seen. Occasional small tubercles without caseation were seen in the fibrous wall. The diagnosis of granulomatous inflammation of the frontal bone consistent with gumma was made. The x-ray examination of the skeleton revealed additional gummas in the left sternoclavicular joint and in the cervical vertebra. The patient did not show signs of emaciation. An antisyphilitic treatment was given.

In this case there were two areas of rarefaction in the frontal bone showing distinct margins and an indistinct honeycomb structure similar to the findings in large hemangiomas of the bone. The involvement of other bones would have been com-

patible with the diagnosis hemangioma. However, the other clinical findings (inflammatory changes of soft tissue, blood test and biopsy results) were definitely in favor of the diagnosis gumma of the frontal bone.

Localized nonspecific periostitis was likewise considered. In large hemangiomas the "sunray pattern" is frequently noticed in the x-ray film. Since this finding is not obtained in common periostitis, a confusion of periostitis and hemangioma is scarcely possible. However, in the presented case the hemangioma was small, and there was no "sunray pattern," and for this reason periostitis came into consideration. The following case shows the difficulty of the differential diagnosis.

R. R., a white man of 38 years, noticed in 1943 severe frontal headache radiating toward the temporal region. He was treated for sinusitis for a long period without success. In 1950 the nasal passages were found to be normal. X-ray examination showed the septum interfrontale bulging toward the left side and a large pneumatic cell in the crista galli. The sinuses were

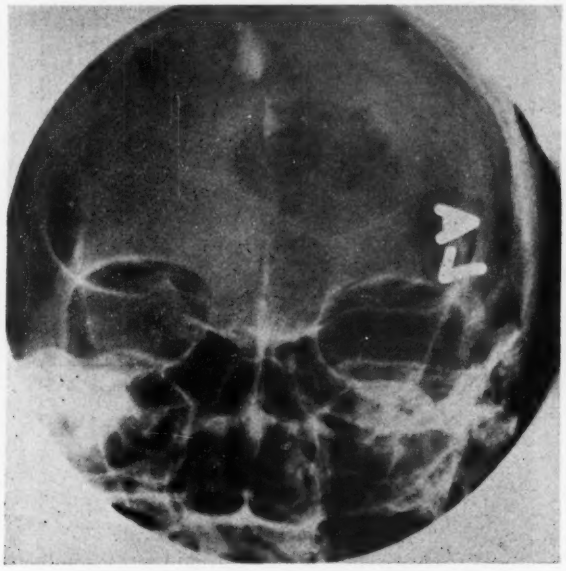

Fig. 3.—Gumma of the frontal bone.

normal. The left frontal sinus showed a large ethmoid cell and a peculiar development of the lateral recessus. Above the left supraorbital arch there was a bony tumor which apparently originated from the anterior wall of the frontal sinus and bulged toward the soft tissue. The surface of the tumor was smooth, and there was no rarefaction of bone (Fig. 4). The tumor was easily palpated. It was not tender, but in that area was an inconspicuous scar. The patient was not aware of the scar and did not know how he received it. The findings did not change for two years. Surgery was refused.

This is a case of traumatic periostitis forming a periosteal osseous tumor on the anterior wall of the left frontal sinus and causing considerable headache. This is a finding not infrequently obtained subsequent to slight injuries of the head. Usually the diagnosis sinusitis is made, and a useless treatment is instituted. In the lateral

exposure the x-ray film showed a finding similar to the finding in the presented case of hemangioma. However, the scar in the skin was in favor of the diagnosis of traumatic periostitis.

CASE 2.—G. F. was a white woman, 28 years of age. Her father died of carcinoma. Six weeks before admission she noticed a small tumor, the size of a cherry, at the right side of the forehead. The tumor grew gradually. It was removed together with the periosteum of the forehead. The microscopic examination revealed osteosarcoma showing cells of chondroid

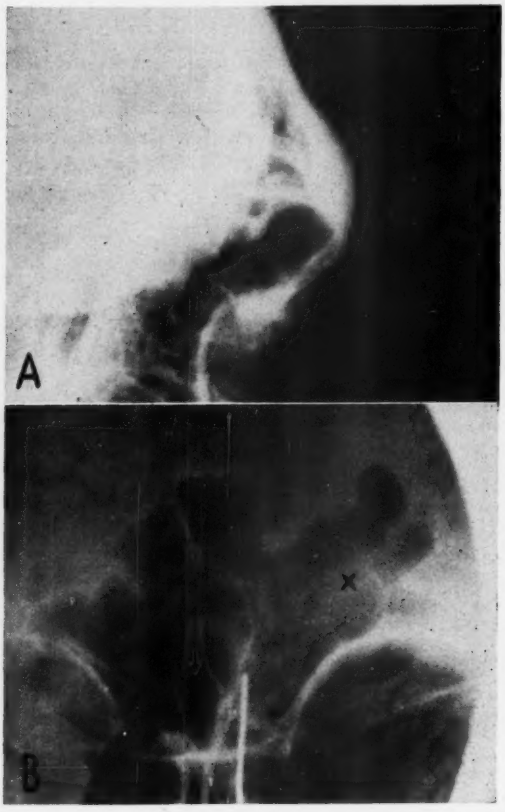

Fig. 4.—*A*, the formation of new bone on the anterior wall of the frontal sinus due to traumatic periostitis. *B*, the arrow indicates the pneumatic cell in the crista galli; *x* indicates a large ethmoid cell bulging into the frontal sinus. Note the deviation of the septum interfrontale and the probe in the left frontal sinus.

type. X-ray films taken on Dec. 30, 1937, revealed cloudiness and indistinct boundaries of the right frontal sinus (Fig. 5). Corresponding to the tumor on the outside, there was a diffuse osseous tumor which bulged into the frontal sinus. There was only a slight bulging of the frontal bone toward the skin. The diagnosis of osteoma or sequestrum subsequent to a frontal sinus infection was made. From Jan. 22, 1938, she complained of slight headache; otherwise, there were no complaints.

At admission, on Jan. 27, 1938, there was a small scar about 5 cm. long, 2.5 cm. above the supraorbital arch on the right side. There was no pathological condition in the nasal passages,

in the lungs, and in the blood. On Jan. 28 an external frontal sinus operation was performed. The anterior wall of the frontal sinus was slightly bulged and was, to a great extent, deprived of periosteum. After removal of the extremely thin anterior wall an anemic white reddish tumor was exposed which had the appearance of pumice stone. The tumor filled apparently the entire sinus; however, it was possible to separate the tumor from the lateral and posterior walls of the sinus. The lateral portion of the posterior wall appeared to be thickened and uneven (*a* in Fig. 6). At this site there were remnants of mucosa and two small openings which led into channels, about 1 cm. long. A probe introduced into these channels touched soft tissue, perhaps dura (*b* in Fig. 6). Another portion of the tumor, the size of a cherry, was separated from the mesial part of the frontal sinus and from the interfrontal septum (*c* in Fig. 6). At this site atrophic mucosa was noticed. It was not possible to probe the nasofrontal duct. For

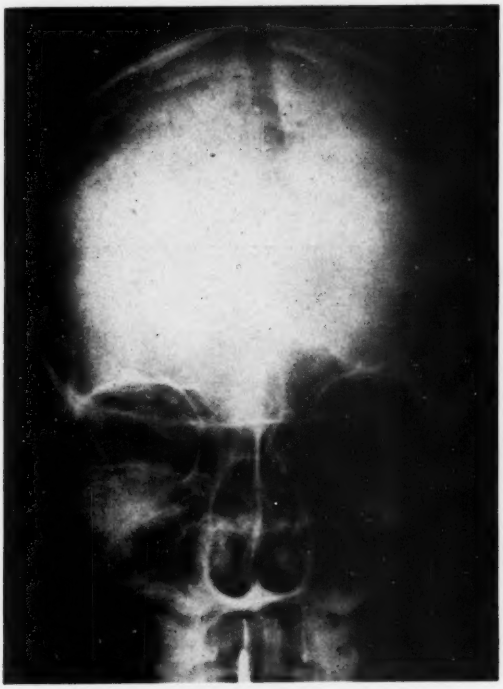

Fig. 5.—Osteogenic sarcoma of the frontal bone.

this reason the frontal process of the maxilla was removed, and the nasal mucosa was incised. But it was not possible to enter the nasofrontal duct from the nostril either, since it was entirely blocked by tumor, which also filled the anterior ethmoid. At the end of the operation the following landmarks could be determined: The mesial portion of the supraorbital arch was removed. The area of the nasofrontal duct and the anterior ethmoid were filled with tumor. The tumor encroached the rest of the supraorbital arch and the interfrontal septum. The tumor likewise replaced a portion of the posterior wall of the sinus. At that site it probably extended toward the dura. A coagulation of the tumor was performed, and the incision was partially closed. After-treatment with x-rays and radium was given. On Sept. 8, 1938, the entire forehead was occupied by tumor masses which apparently were localized subperiosteally. A short time later the patient died. No autopsy was performed.

Microscopic Examination.—The bulk of the tumor consisted of polyhedral cells with hyperchromatic nuclei and poorly defined cell borders (malignant osteoblasts). There was no pleo-

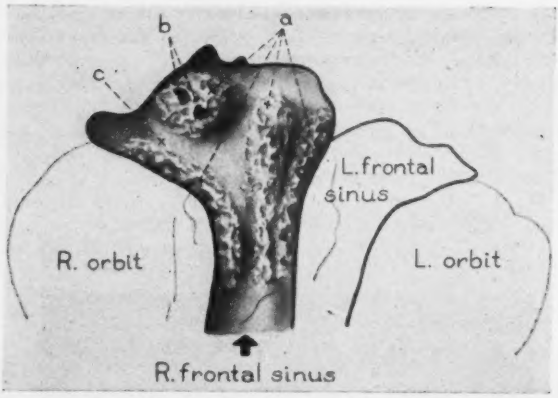

Fig. 6.—Surgical finding in a case of osteogenic sarcoma. The picture shows the posterior wall of the right frontal sinus: *a*, uneven bone grown together with the tumor; *b*, fistulae leading toward the dura; *c*, normal mucosa.

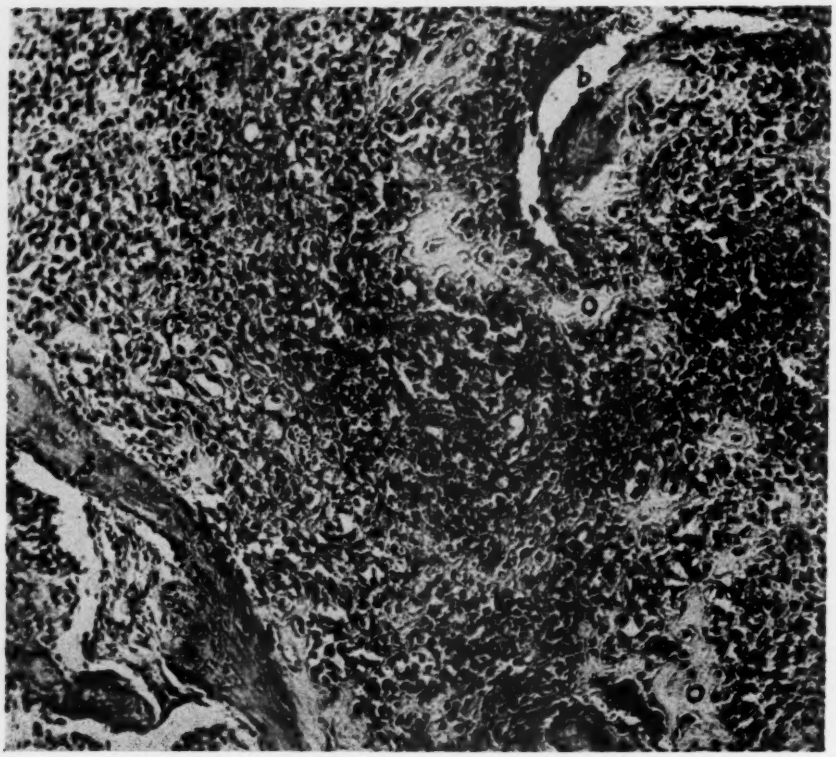

Fig. 7.—Osteogenic sarcoma of the frontal bone: *b*, blood space rimmed by malignant osteoblasts; *o*, osteoid trabeculae; *s*, spicule of normal reactive bone.

morphism of the cells. There was formation of a moderate amount of osteoid substance (*o* in Fig. 7). The osteoid formed spicules with indistinct margins. On other places the tissue had a chondroid appearance (Fig. 8). There was only a small amount of distinct blood vessels, but there were small blood spaces rimmed by malignant osteoblasts (*b* in Fig. 7). There were spot-like calcium deposits.

This was an osteogenic sarcoma of sclerosing type involving the frontal squama. The bulk of the tumor consisted of malignant osteoblasts producing a fair amount of osteoid substance. There was, in addition, a considerable amount of calcium deposits in the tissue. On some places the tissue had the appearance of atypical cartilage.

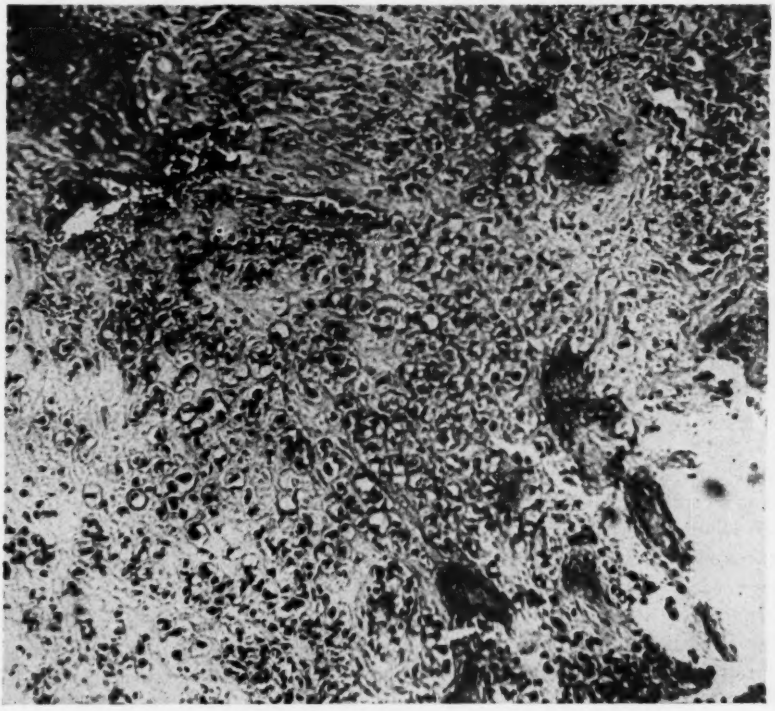

Fig. 8.—Osteogenic sarcoma. Cartilage-like tissue; *c*, calcium deposits.

According to Geschickter and Copeland,[10] these tumors are commonest about the knee and take their origin in the osteogenic layer of the periosteum or the endosteum. Osteogenic sarcoma of the cranial bones is rare. In a series of more than 700 cases of primary bone sarcomas the cranial bones were involved in only 12, and in these cases the parietal, occipital, and temporal bones were affected but not the frontal bones (Geschickter and Copeland). In the presented case the tumor caused an obliteration of the frontal and ethmoid sinuses on the right side.

10. Geschickter, C. F., and Copeland, M. M.: Tumors of the Bone, Ed. 3, Philadelphia, J. B. Lippincott Company, 1949.

However, the tumor did not take its origin from the ethmoid, although sarcomas of the ethmoid occur more frequently than sarcomas of the frontal sinus. In three cases of sarcoma of the ethmoid reported by me [11] the tumor was visible in the nasal cavity and caused orbital symptoms. In none of these cases did the tumor grow into the frontal sinus. In the presented case the tumor, being in the advanced stage, was not visible in the nasal cavity and there were no orbital symptoms, but there was an involvement practically of the entire frontal squama. This indicated that the tumor did not take its origin in the ethmoid but extended secondarily into this cavity.

Since the paranasal mucosa serves as periosteum, the question arises as to whether the tumor took its origin from the mucosa of the frontal sinus. The question must be answered in the negative. This statement is proved by the following findings: (1) The small tumor of the forehead which was removed for biopsy was undoubtedly of periosteal origin, and (2) the mucosa of the frontal sinus was normal to a considerable extent. These findings indicate that the tumor did not take its origin in the frontal mucosa but in the periosteum (and endosteum) of the frontal squama. In other words, this was not a tumor of the frontal sinus but a tumor of the frontal squama extending into the frontal and ethmoid sinuses.

With this diagnosis the admixture of cartilage-like areas in both the tumor of the forehead and the tumor in the frontal sinus is difficult to understand because the histological nature of osteogenic sarcoma is supposed to repeat in all of its essentials the normal formation of bone. For this reason cartilage-like areas may occur in osteogenic sarcomas of the tibia or the femur because these bones develop from a cartilaginous anlage. However, the frontal squama develops by direct ossification of connective tissue without passing a stage of cartilage; thus, cartilage-like areas in sarcoma of the frontal squama cannot be explained by a duplication of embryological development. One must rather assume that in the presented case, under the influence exerted by the tumor, the periosteum had lost its specific activity as a center of bone growth and had acquired a polyvalent function by producing both bone and cartilage. This is what likewise occurs in embryological life. The periosteum of human embryos may lay down bone and, immediately next to it, cartilage which is called "secondary cartilage." This condition occurs in humans during the ossification of the mandible and the clavicle. In animals it occurs during the ossification of the hard palate, vomer, pterygoid bone, malar bone, and other bones (Schaffer). The mature periosteum lacks the polyvalent function, but it may reacquire it under the influence of osteogenic sarcoma.

In the presented case the patient was 28 years old. This age corresponds with the upper limit of the age group of patients suffering from osteogenic sarcoma of the lower extremity. While the duration of osteosarcoma of skeletal bones averages under 10 months (Geschickter and Copeland), no information is available in the presented case in this respect. The first symptom caused by the neoplasm was a small tumor of the forehead. But at that time the sarcoma was far-advanced. Pain was not marked; headache was not conspicuous. The prominent symptom was diffuse sclerosis of the frontal squama and the frontal sinus. This is a common

11. Brunner, H.: Zur Klinik maligner Tumoren in der ethmo-orbitalen Region, Monatsschr. Ohrenh. **70**:129, 1936.

12. Footnote deleted.

symptom in osteogenic sarcoma of the skeletal bones. Geschickter and Copeland claim that the sclerosis is due to a combination of both tumor bone and reactive bone; the formation of new bone "is generally less in amount than the subperiosteal growth, but in rare instances may keep pace with it." In the presented case the sclerosis was more evident than the subperiosteal tumor growth. For this reason the most striking x-ray finding was the formation of a tremendous amount of new bone in the diploë and the involved sinuses, obliterating the normal osseous markings and the cavities of the involved sinuses.

This finding is in sharp contrast to the findings in primary carcinoma of the frontal sinus in which destruction of bone prevails. The following case is reported to demonstrate the contrast in the x-ray findings in primary carcinoma of the frontal sinus on one hand and in osteogenic sarcoma of the frontal bone on the other hand.

Fig. 9.—Primary carcinoma of the frontal sinuses.

J. B., a white man of 40 years, on Aug. 5, 1943, while shaving, noticed a hard lump on the ridge of the nose between the left eye and the nose. The skin over the tumor was normal. The mass gradually became larger. In the last 2½ months prior to admission he had every morning on rising a sharp pain lasting for one-half hour right over the swelling. For the last three weeks there was loss of smell. There were no headache, no visual disturbances, no discharge from the nose, no history of injury and no tenderness. He complained of difficulty in breathing.

At admission, on Nov. 1, 1943, there was a large swelling at the nasal ridge. The nasal passages were normal, except for a deviation of the septum to the right. The posterior wall of the pharynx was rough, with bloody postnasal drip. X-ray showed a large defect, with distinct margins in the area of the frontal sinuses (Fig. 9). The lungs were clear.

A curvilinear incision was made along the lower margin of each brow meeting in the midline at the base of the nose. The tumor mass was mobilized and on removal broke off at the ethmoid region. Microscopic examination revealed a nonhornifying squamous-cell carcinoma. Radium seeds were implanted. Nevertheless, in January, 1944, a metastatic hornifying squamous-cell carcinoma of the bronchus was discovered which it was decided was inoperable.

Primary carcinomas of the frontal sinus are rare. Like sarcoma of the frontal bone, carcinoma of the frontal sinus has a latent course for a long period, and nasal symptoms are absent most of the time. In the presented case there was a bloody postnasal drip, but this occurred in the advanced stage of the disease. The differential diagnosis was made on the basis of the x-ray film: destruction of bone without formation of reactive bone in carcinoma and diffuse sclerosis of bone in osteogenic sarcoma.

However, the finding of diffuse sclerosis per se does not permit the diagnosis of osteogenic sarcoma, because the same or a similar finding may be obtained in fibrous dysplasia, Paget's disease, and, particularly, in chronic osteomyelitis of the frontal bone. From a practical point of view chronic osteomyelitis of primary or secondary type (Brunner [13]) is most interesting because it is common and is frequently not properly evaluated by both rhinologist and radiologist. For this reason a case of secondary chronic osteomyelitis is reported because it shows the progressive densification of the bone, analogous to the finding in osteogenic sarcoma, to an extreme degree. In Sept. 10, 1945, the case history was reported in another place.[13a]

M. P. was a 21-year-old white woman. In 1939 acute bilateral frontal sinusitis developed after swimming, which was followed by acute osteomyelitis. No radical operation was performed, but several sequestra were removed from the skull on both sides. In 1940 a draining sinus developed above the lateral angle of the left eye. Sequestra were removed from that area until January 1942. On March 28, 1942, the patient was examined by me for the first time. The Wassermann test in the blood gave negative results. There was a draining fistula in the outer angle of the left eye and a moderate loss of hearing on the right side. An x-ray film taken on Aug. 3, 1942, revealed both frontal sinuses to be cloudy and surrounded by sclerotic bone. The boundaries of the left frontal sinus were indistinct. The sclerosis of the bone extended toward the left malar bone and the left ala magna of the sphenoid, narrowing the orbital fissure on that side. In several places the diploë and the external lamina of the bone were replaced by newly formed sclerotic bone. The internal lamina was intact. The posterior wall of the frontal sinus and the roof of the orbit were thickened. Since the patient complained of pain in the right leg, an x-ray film was taken; it showed a spindle-like thickening of the cortex of the right tibia, with narrowing of the central marrow space. Within the thickened area there was a spot of translucency. This was interpreted as metastatic osteomyelitis of the right tibia. On Aug. 8, 1942, a radical frontal operation was performed on the left side. There was granulating periostitis, and the floor of the sinus was thickened. The frontal mucosa was normal. From the draining fistula at the lateral angle of the eye a channel, filled with granulations, ran along the thickened malar bone 1.5 cm. into the depth of the orbit. After the operation the draining fistula became permanently closed, and the patient felt well.

On Sept. 10, 1945, she suddenly noticed a fulness and a diminution of hearing on the left side. On Sept. 14, 1945, the deafness increased on both sides. On Sept. 15 she noticed that "something snapped" in the left ear and that suddenly she became entirely deaf on the left side. On Aug. 4, 1948, she was totally deaf on both sides. The drums were normal, and the labyrinths were excitable on both sides. X-ray of the temporal bones failed to show pathological conditions. An x-ray film, taken on Aug. 28, 1948, revealed that the sclerosis of the bone had progressed considerably in the frontal squama, in the malar bone, and particularly in the roof of the orbit (Fig. 10).

The predominant x-ray feature in this case was progressive sclerosis and thickening of certain bones of the skull causing total deafness on both sides. The pathogenesis of the deafness is not clear, but evidently there is a relationship

13. (*a*) Brunner, H.: Chronic Osteomyelitis of the Skull, Ann. Otol. Rhin. & Laryng. **52**:850, 1943; (*b*) Self-Limited Osteomyelitis of the Skull and the Paranasal Sinuses, J. Laryng. & Otol. **65**:575, 1951.

between deafness and chronic osteomyelitis. The progressive sclerosis was not caused by a syphilitic infection or by osteogenic sarcoma or by fibrous dysplasia. The long duration of the disease, the finding of granulations, sequestra, and draining fistulae, and the negative Wassermann test results were not in favor of these diagnoses. For this reason the diagnosis was chronic osteomyelitis of secondary type, viz., subsequent to acute osteomyelitis of the frontal bone.

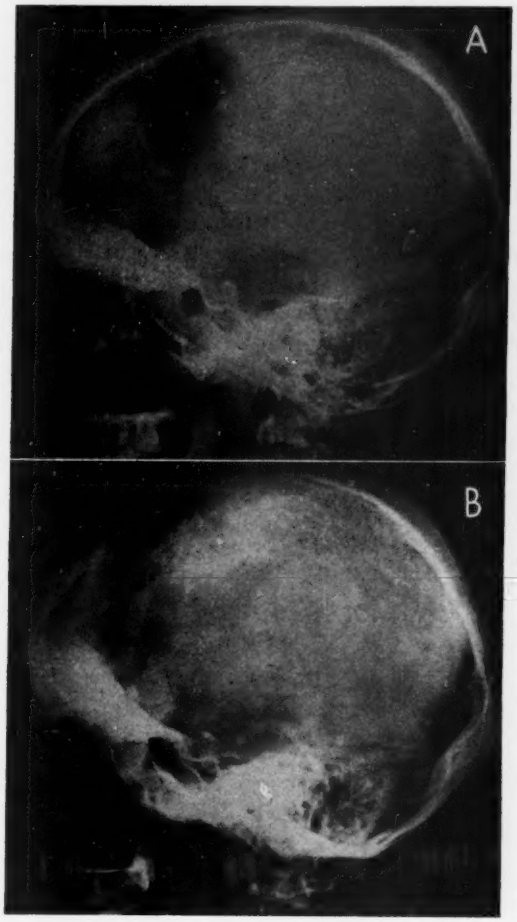

Fig. 10.—In *A* the picture was taken on Aug. 3, 1942, and in *B* on Aug. 28, 1948. Replacement of diploë and external lamina by newly formed sclerotic bone (*x*). Note the increasing sclerosis and thickness of the frontal squama and the roof of the orbit.

In contrast to osteogenic sarcoma, the following microscopical findings are significant of chronic osteomyelitis: The osteoblasts are not formed in excess, and they are lined in orderly rows about bone spicules; the osteoid tissue bears constant relationship to the amount of fibrous tissue or the number of osteoblasts present; the infiltration with leucocytes and the formation of granulations are the predomi-

nant findings, the latter serving in resorption of both original and newly formed bone. These findings assist in the differential diagnosis between chronic osteomyelitis and osteogenic sarcoma of the frontal bone.

SUMMARY AND CONCLUSIONS

Tumors arising from the mucosa of the frontal sinus occur less frequently than tumors of the ethmoid and maxillary sinuses. Among other causes there are two factors which may help to explain the difference: The frontal sinus is absent in approximately 5 to 10% of normal adults, and tumors of the frontal sinus cause nasal symptoms much later than tumors of the ethmoid or the maxillary sinuses.

A case of small hemangioma of the frontal bone, apparently arising from the diploic veins, is reported, and the differential diagnosis is discussed between hemangioma on one hand and syphilitic periostitis and nonspecific periostitis of the frontal bone on the other hand. The hemangioma was surgically removed without laying free the dura. There was no recurrence in a period of 10 years.

A case of osteogenic sarcoma of sclerosing type arising from the periosteum of the frontal squama is reported causing an obliteration of the frontal sinus. The differential diagnosis is discussed between osteogenic sarcoma on one hand and primary carcinoma of the frontal sinus and chronic osteomyelitis on the other hand.

The cartilage-like areas in the osteogenic sarcoma are explained by a reappearance of the embryologic function of the periosteum.

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LARYNGOFISSURE APPROACH IN SURGICAL TREATMENT OF BILATERAL ABDUCTOR PARALYSIS

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THERE have been many approaches to the surgical treatment of bilateral abductor paralysis. King,[1] Kelly,[2] Orton,[3] Woodman,[4] and Thornell[5] have their own procedures which are satisfactory in their hands. All their procedures have the same objectives: (1) to reestablish an adequate airway and (2) to obtain and maintain the maximum speaking voice, without sacrificing the airway. The attainment of these objectives, each to its maximum degree of efficiency, has not been achieved with any real ease or calculation. It is the purpose of this paper to suggest a new simple laryngofissure procedure for the surgical treatment of bilateral abductor paralysis.

The technical difficulties entailed in either the Kelly or the King operation cannot be overlooked. They are very tedious procedures, and the visual exposures obtained tend to make the surgery still more difficult. In each procedure a visual estimate is made of the size of the glottic chink, so that an adequate airway and voice can be obtained. Woodman[6] has shown that "a space of 3 to 4 mm. is adequate. Such a space always results in a good speaking voice. If the space is wider than 6 mm.. the quality of the voice suffers."

In most of the procedures that have been done in the past, whether or not the arytenoid is removed, the vocal cord is sutured in abduction at the time of the surgery. Once the vocal cord is fixed, its position cannot be adjusted later if necessary. This result has several drawbacks. The size of the glottis may need to

Read at the meeting of the International College of Surgeons, Section on Otolaryngology, Sept. 3, 1952, Chicago.

1. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis: Preliminary Report, J. A. M. A. **112**:814-823 (March 4) 1939.

2. Kelly, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles, Arch. Otolaryng. **33**:293-304 (Feb.) 1941.

3. Orton, H. B.: Extralaryngeal Surgical Approach for Arytenoidectomy: Bilateral Abductor Paralysis of the Larynx, Ann. Otol. Rhin. & Laryng. **53**:303-307 (June) 1944.

4. Woodman, DeG.: A Modification of the Extralaryngeal Approach to Arytenoidectomy for Bilateral Abductor Paralysis, Arch. Otolaryng. **43**:63-65 (Jan.) 1946.

5. Thornell, W. C.: New Intralaryngeal Approach in Arytenoidectomy in Bilateral Abductor Paralysis of Vocal Cords: Report of 3 Cases, Tr. Am. Acad. Ophth. **53**:631-636 (July-Aug.) 1949.

6. Woodman, DeG.: The Open Approach to Arytenoidectomy for Bilateral Abductor Paralysis, with a Report of 23 Cases, Ann. Otol. Rhin. & Laryng. **57**:695-704 (Sept.) 1948.

be wider in certain persons if they have an active occupation. A more sedentary person may get along well with a narrower glottic chink and hence be made to have a better voice. This factor should be considered in the over-all picture.

After the fixation of the vocal cord there may be some secondary change in position following healing, with resultant scar formation. (It is difficult to estimate this amount of change in position of the vocal cord at the time of the surgery.)

Easy visualization of the operative field is certainly desirable. Yet, all the external procedures done today have only fair visualization, and so the procedure becomes a more tedious one. Certainly the ideal surgical procedure for bilateral abductor paralysis would be one that included the following features: (1) direct visualization of the operative field; (2) surgery that would reestablish an adequate airway for any person, and (3) result in the maximum speaking voice. With these desirable features in mind I devised such an operation which would satisfy these requirements.

The most direct approach to the vocal cords and arytenoids is via the laryngofissure technique. Dr. Thomas Edwards [7] in his review of the literature on bilateral abductor paralysis described what had already been done via this approach. Certainly this is the easiest way to visualize the arytenoids and hence the simplest way to do an arytenoidectomy. On the cadaver I found it more difficult to do the arytenoidectomy by the posterior approach or through a window in the thyroid cartilage. The reason for the removal of the arytenoid is to give more space in the posterior portion of the glottic chink once the vocal cord has been put into a position of abduction.

The question of intralaryngeal scarring and contraction arises after the removal of the arytenoid cartilage via the laryngofissure approach. However, if the mucosal edges of the bed of the arytenoid are approximated surgically, this danger of contraction and encroachment on the laryngeal aperture can be eliminated. The preoperative and postoperative use of the antibiotics will prevent other complications.

I originally thought that the suturing back of the vocal cord to the thyroid cartilage at the time of the arytenoidectomy was not desirable. The idea at the onset was that it is preferable to have complete intralaryngeal healing of the arytenoid bed and disappearance of all edema before the cord is put into abduction. Only in this way can one accurately determine the final position of the vocal cord. Whatever scarring takes place certainly will change the position of the vocal cord, so that one month later there is either a larger or a smaller glottic chink than was calculated at the time of the surgery. In the procedure as first done by me I tried to eliminate this visual approximation. At the time of the arytenoidectomy a stainless-steel suture is placed through the vocal cord and brought out through the thyroid cartilage and a small second incision on the side of the neck. The vocal cord is allowed to remain in its original paralyzed position, and the larynx is closed and left to heal for two weeks without disturbance. The second incision down to the perichondrium of the thyroid cartilage, where the suture issues, is kept open with aureomycin packing. In two weeks all the laryngeal edema subsides, and the vocal cords can be seen on indirect examination. The second stage of the procedure is then performed.

7. Edwards, T. M.: Progress in the Surgical Treatment of Bilateral Laryngeal Paralysis, Ann. Otol. Rhin. & Laryng. **61**:159-178 (March) 1952.

With the use of local anesthesia the vocal cord can be adjusted by the stainless-steel suture which has been brought out laterally on the neck at the time of the original procedure. It is like working a very stiff puppet. The vocal cord can be adjusted to a suitable point of abduction in order to obtain a good airway, at the same time correlating this with the voice of the patient. The patient is allowed to breathe deeply and to speak during this adjustment. Having determined the amount of pull necessary on the single suture to abduct the vocal cord sufficiently, the surgeon fixes it at that point to the thyroid cartilage. The second skin incision is then closed. Indirect laryngeal examination at this time will show the exact position of the vocal cord.

This two-stage procedure seemed at first like the most ideal operation for bilateral abductor paralysis to obtain the precise position in which the vocal cord should be placed to give an adequate airway and optimum voice. However, certain factors make this second stage dangerous and difficult. During the course of keeping the second incision open for two weeks there is a certain amount of filling in underneath the packing and epithelization of the wound edges. This granulation on the surface of the thyroid cartilage will obscure the exits of the stainless-steel suture. The amount of pull needed on the suture to abduct the vocal cord is greatly increased during this two-week waiting period. At the first stage a very gentle pull brings the cord into abduction, whereas at the second stage the tension on the suture has to be greatly increased. There is also increased difficulty in accurately tying the suture on the thyroid cartilage deep in the constricted wound while exerting the necessary pull. Since the entire operation depends on complete accuracy in fixing the single suture during the second stage, the risk of failure at this time is too great to warrant undertaking a two-stage operation. The completion of this operation at one stage thus seems like the logical answer. At this time the suture can be accurately tied in place over the thyroid cartilage and the vocal cord abducted according to a visual approximation of the required airway.

I have taken the liberty of reporting this early only because of the surgical simplicity of the procedure and the good result obtained. Having tried the various operations for bilateral abductor paralysis on many cadavers, I feel that the technique which I shall describe is the easiest and most direct approach to the problem.

DESCRIPTION OF THE SCHEER OPERATION

The entire operation can be done with the use of local anesthesia. A preliminary tracheotomy must be done (if it has not already been done) by extending the midline incision.

Tetracaine (pontocaine®) hydrochloride, 2%, is instilled into the larynx under indirect vision. The neck is injected with 2% procaine hydrochloride. Since most patients will already have a tracheotomy tube in place, the midline incision is made from the thyroid notch down to within 3 mm. of the tracheotomy tube. The ribbon muscles are separated in the midline, and the thyroid cartilage is exposed. The cricothyroid membrane is exposed. A horizontal incision is made in the cricothyroid membrane after a few drops of 10% cocaine solution have been instilled through the membrane into the trachea. Through this opening ½ in. (1.3 cm.) packing is put in place down on to the tracheotomy tube. A laryngofissure scissors is used to split the thyroid lamina, care being taken that the blade of the shears is between the paralyzed vocal cords. The larynx is then opened and held apart with two dull

retractors. Cocaine, 10%, is sprayed into the larynx and into the pyriform sinuses. A no. 3-0 stainless-steel suture on a small cutting edge needle is put through the vocal cord about 2 mm. in front of the vocal process of the selected vocal cord. The

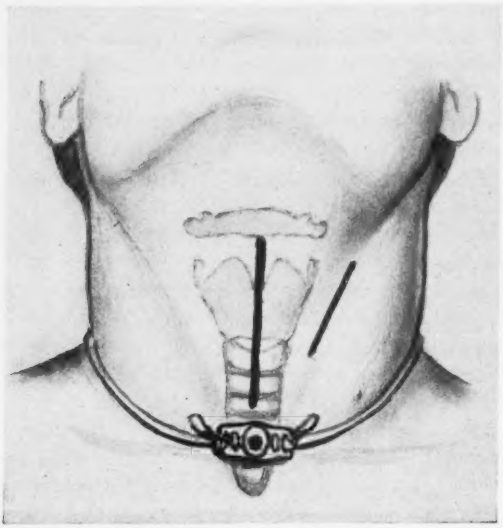

Fig. 1.—The two incisions used in the Scheer operation for bilateral abductor paralysis.

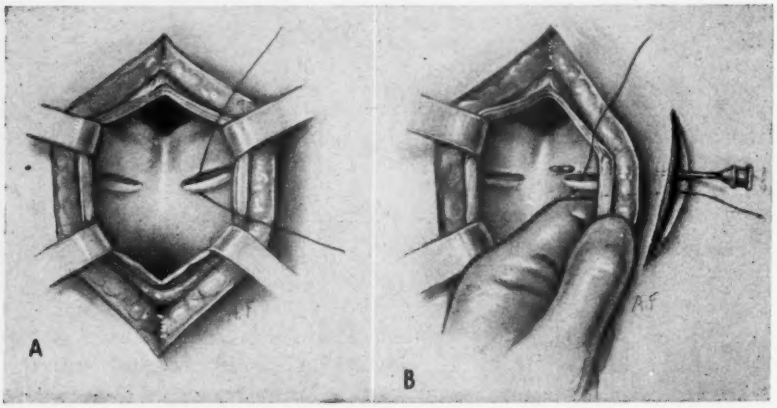

Fig. 2.—*A*, stainless-steel suture in place through vocal cord, just distal to the vocal process. *B*, a 17-gauge needle entering the larynx through the second incision. The point of the needle penetrates the laryngeal mucosa next to the steel suture where it comes through the vocal cord. This step is repeated above and below the vocal cord.

suture is placed from below upwards just beneath the mucosal surface of the vocal cord. The needle is removed, and the suture remains in place with both ends free (Fig. 2 *A*).

An incision is made on the same side of the neck in the direction of the sternomastoid muscle extending the length of the thyroid cartilage starting ¼ in. (0.6 cm.) anterior to the posterior margin of the thyroid lamina (Fig. 1). The fibers

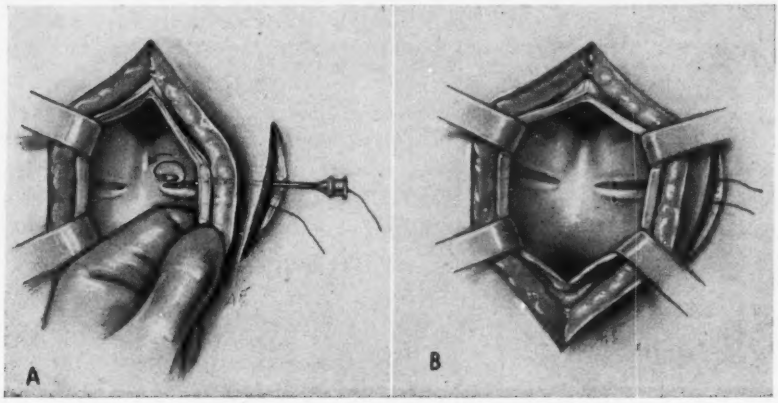

Fig. 3.—*A*, the free ends of the steel sutures are fed into and through the needle, which is then withdrawn. *B*, the single suture is in place, having been drawn through the lamina of the thyroid cartilage.

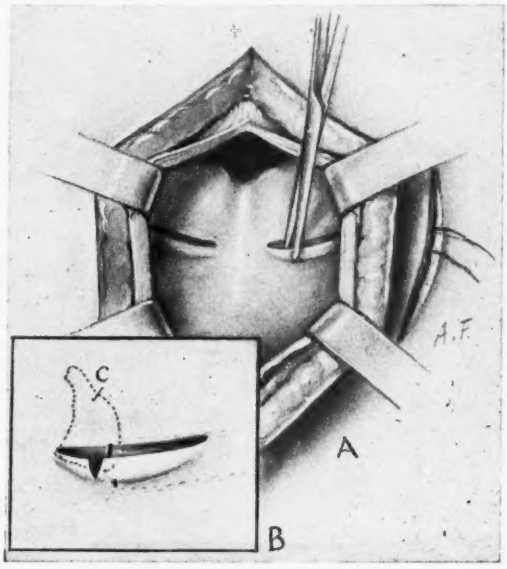

Fig. 4.—Scissors ready to cut the vocal cord (*A*). Insert (*B*) indicates that the vocal process of the arytenoid (*C*) has been cut through just proximal to the stainless-steel suture.

of the sternothyroid muscles are separated by blunt dissection to expose the perichondrium of the thyroid cartilage along its length ¼ in. from its posterior edge.

While the surgeon looks into the larynx to get the correct position, a 17-gauge needle is inserted through the outer lamina of the cartilage to extend inward to

the area where the suture through the vocal cord is placed. The needle is inserted on a line with the vocal cord but slightly posterior and superior to the vocal process. Therefore the needle is angulated anteriorly and medially on its insertion. The needle is pushed inward slowly until its point becomes visible just above the vocal

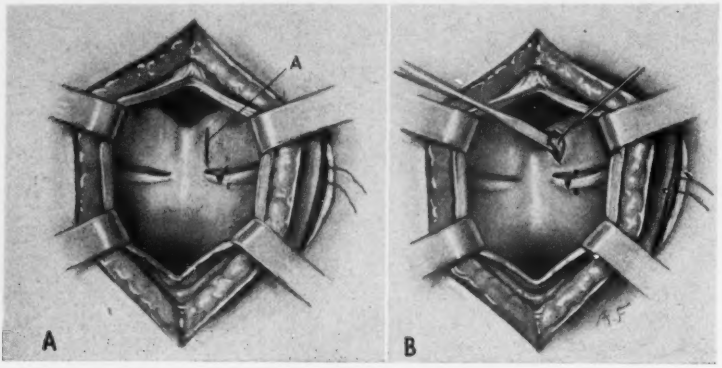

Fig. 5.—*A*, vertical incision (*A*) through the laryngeal mucosa over the arytenoid. *B*, the arytenoid is fixed with a sharp hook while subperichondrial dissection is being carried out. The arytenoid is then removed.

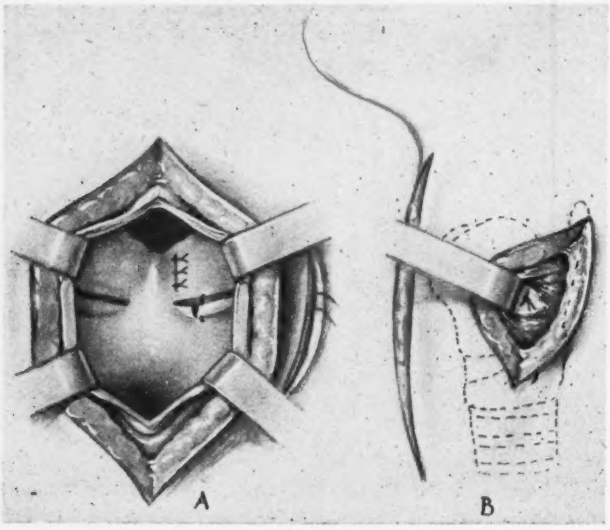

Fig. 6.—*A* shows the arytenoid bed closed with interrupted sutures. *B* shows the stainless-steel suture tied over the thyroid cartilage after it had been tightened sufficiently to place the vocal cord in its proper degree of abduction.

cord where the suture has been placed. The point of the needle is allowed to break through the mucosal surface next to the stainless-steel suture (Fig. 2 *B*). The upper free end of the suture is fed into the open end of the needle until it comes out of its proximal end (Fig. 3 *A*). The needle is then withdrawn, allowing the

free end of the suture to remain outside of the thyroid cartilage. The same step is repeated with the 17-gauge needle except that its point of insertion on the thyroid lamina is ¼ in. inferior to the first opening but on the same line parallel with the posterior border of the thyroid cartilage. This time the lower free end of the suture within the larynx is threaded into the needle and brought out externally, and the needle is removed (Fig. 3 *B*).

With sharp-angled scissors the vocal process is cut through close to the body of the arytenoid cartilage (Fig. 4). The arytenoid is now ready to be removed. A small quantity of procaine hydrochloride with epinephrine is injected into the arytenoid area with an attempt to get some subperichondrially. A vertical incision is made on the anterior surface of the arytenoid cartilage from the cricoarytenoid joint to the upper pole of the arytenoid (Fig. 5 *A*). By subperichondrial dissection

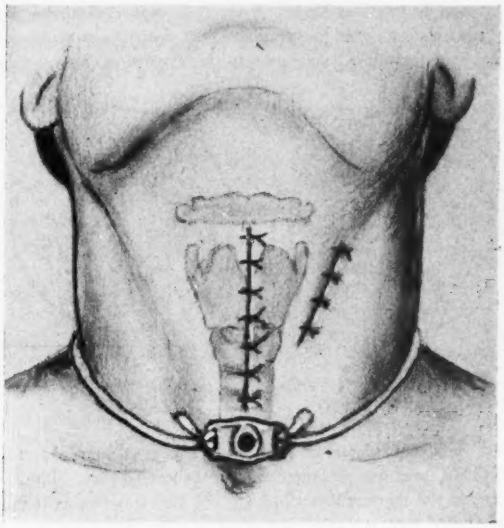

Fig. 7.—Both external incisions closed.

the cricoarytenoid joint is disarticulated, and the entire arytenoid is removed subperichondrially (Fig. 5 *B*). There is a minimum amount of bleeding during the removal of the arytenoid. On its completion the bed of the arytenoid is noted to be laid wide open. No. 3-0 plain sutures on a small sharp cutting needle are used to close the bed of the arytenoid. The mucosal edges are brought together in the original vertical line of the incision (Fig. 6 *A*). The interrupted sutures incorporate the muscular bed of the arytenoid to prevent any dead space. Approximately three sutures are necessary.

By a gentle pull on the external stainless-steel suture the vocal cord is now noted to move in abduction. A visual estimate of the amount of abduction necessary is made by the surgeon, and the free ends of the steel suture are tied down on the thyroid cartilage, thus fixing the vocal cord in abduction (Fig. 6 *B*). This procedure is done under direct vision by the operator. The laminae of the thyroid

cartilage are released and the ribbon muscles brought together with interrupted chromic sutures, and the skin of both incisions is closed with interrupted silk sutures (Fig. 7). The tracheotomy tube is left in place.

REPORT OF A CASE

The patient, a white man aged 64, was admitted to the University Hospital on Nov. 22, 1950. At that time there were bilateral fixed masses in the thyroid. A total thyroidectomy was done. The pathological examination revealed adenocarcinoma of the right lobe and papillary adenocarcinoma of the left lobe of the thyroid. A tracheotomy was done at the time of the surgery. While he was in the hospital, x-rays of the vertebrae showed metastases to the fourth and fifth lumbar vertebrae. On discharge from the hospital he was given a course of radiation to the fourth and fifth vertebrae. There was considerable improvement in the roentgenographic picture, and the patient gained weight. The patient was a diabetic, regulated on insulin for 10 years.

The patient had the tracheotomy tube in place for 17 months, during which time he was susceptible to frequent respiratory infections. If the tube was corked, he would absolutely be unable to inspire any air. He was able to speak in short deep raspy sentences. A recording of his voice was made before operation to demonstrate this condition. Indirect laryngeal examination showed complete bilateral abductor paralysis. He was admitted to the Misericordia Hospital on May 15, 1952, and was operated on the following day. The operation was done on the right vocal cord in the manner described for the two-stage procedure. When the larynx was split open, it was noted that the left vocal cord overlay the right in such a manner as to explain the complete inability for inspiration. One-half hour following surgery the patient was able to swallow water taken from a drinking tube. He was given penicillin daily for seven days. A soft diet was started the day after surgery. There was some pain on swallowing which completely disappeared by the sixth day. The dressing was changed on the fifth day, at which time the larynx was examined with a laryngeal mirror. There was marked edema of the epiglottis and both arytenoids, particularly the right arytenoid area. The sutures from the midline incision were removed on the seventh day. The second incision was still open, and the aureomycin packing was in place. There was no sign of infection. On the 14th day there was no laryngeal edema, so that the second stage was done. The difficulties involved in this stage have already been discussed. The tracheotomy tube was corked at the time of the second stage, and the patient was able to breath comfortably without the use of the tracheotomy tube for the first time since November, 1950. During the course of the next four days gradual difficulty in breathing again became apparent, and the cork had to be removed. Indirect laryngeal examination now revealed that the left vocal cord, which was noted to be beyond the midline at the time of the first operation, had moved still farther toward the right. There was also incomplete abduction of the right vocal cord because of the difficulties met with in the second stage.

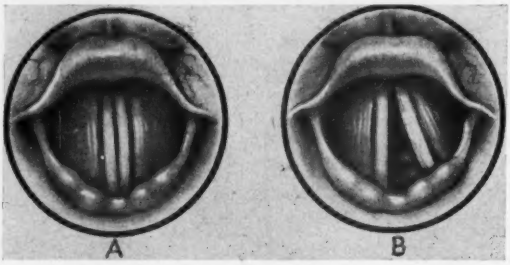

Fig. 8.—*A* shows both vocal cords paralyzed in mid-line position. *B* shows the left vocal cord fixed in abduction following surgery.

On June 16, 1952, the larynx was again opened, and the one-stage procedure was done on the left vocal cord. The ease and accuracy of adjusting the cord in abduction have been described. The tracheotomy tube was again corked, and the patient was discharged from the hospital six days after the operation. At the end of the second week there was only a minimum amount of laryngeal edema. The glottic chink was noted to be about 4 to 5 mm. at its posterior point. On July 10, a little over three weeks after the left vocal cord was surgically abducted, the tracheotomy tube was removed, and the tracheal stoma was closed. The patient has returned to work and has resumed all his normal activities. The excessive bronchial secretion which was so profuse before surgery has completely disappeared.

SUMMARY

A laryngofissure approach for arytenoidectomy is described in the treatment of bilateral abductor paralysis. A two-stage procedure has been tried, with the idea of increasing the accuracy in the final determination of the degree of abduction of the vocal cord. The two-stage procedure was not found to be practical in my hands. The simpler one-stage procedure has been adopted. The decided advantage of this approach is the direct visualization of the surgical field and the simplicity of the entire procedure in fixing the vocal cord in abduction. One case is reported in which a most satisfactory result was obtained.

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SUBCUTANEOUS RUPTURE OF THE TRACHEA

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SUBCUTANEOUS rupture of the trachea is that type of injury in which there is a tracheal tear (varying in size from 1 mm. to complete transection) without an accompanying break in the overlying skin. Because the perforation is limited to the trachea, air escapes into the surrounding tissues but cannot pass to the outside. Lacking an external exit, this air then travels along fascial planes upward and downward to produce numerous problems in diagnosis and treatment.

The following cases are presented to illustrate the occurrence of subcutaneous injuries of the trachea in children, with two divergent but equally successful plans of therapy.

REPORT OF CASES

CASE 1.—T. F., a 6-year-old white girl, was apparently well until she was pushed against an iron fence, receiving a blow across the front of the neck. She subsequently felt pain, tenderness, and a slight swelling in the region of trauma but continued her normal school activities the rest of the day. When she reached home at 5:00 p. m., about six hours after receiving the injury, she complained of pain in the neck. She then had a coughing spell. After this there rapidly appeared swelling of the neck and surrounding tissues, so that in approximately 10 minutes there was obvious bilateral facial deformity and swelling of the shoulders, arms, and chest.

A physician who was seen one-half hour later gave ½ grain (30 mg.) of phenobarbital but no other treatment. She was then brought to Childrens Hospital, Los Angeles, where she was first seen by me, at 9:30 p. m. Close questioning at that time revealed that there had been no noticeable increase in swelling after the initial 10-minute period.

Examination on admission revealed obvious deformity of the face and neck, worse on the left side, with swelling extending superiorly to the origin of the temporal muscle. The left eye was closed by a large swollen upper lid. Swelling involved also both shoulders, arms, and chest, front and back. In all these areas subcutaneous crepitus was easily felt. The child had shallow rapid respirations but lay quietly and was apprehensive.

Over the anterior neck in the region of the second and third tracheal rings there was a bluish skin discoloration several centimeters long running obliquely over the trachea from left superiorly to right inferiorly. This was assumed to be the site of trauma. Palpation of the trachea was not possible because of the distorted overlying soft tissues.

The voice was of good intensity and quality. There was no stridor or retracting. The heart border could not be outlined by percussion. The point of maximum impulse was indistinct, and the tones were distant. There was no audible extraneous noise (Hamman's sign).

On admission the temperature was 98.6 F., pulse rate 104 beats per minute, respiration rate 22, and blood pressure 112/68. Admission x-ray showed that there was marked cervical, thoracic, and mediastinal emphysema. The lung fields were clear, and there was no sign of pneumothorax.

Because the patient was not greatly distressed and there was no obvious progression of signs or symptoms in the four hours prior to admission, a conservative plan of treatment was chosen.

From the Department of Surgery (Otolaryngology), University of Southern California, and the Department of Otolaryngology, Childrens Hospital, Los Angeles.

She was kept at bed rest, with a special nurse and physician in constant attendance. A tracheotomy set and a set for pleural or mediastinal aspiration were in the room for immediate use. Blood pressure, pulse rate, and respiration rate were checked every 20 minutes for the first eight hours. During this period the pressure remained at roughly 110/70, the pulse rate at 100 to 110 beats per minute, and the respiration rate at 24 to 28.

A x-ray picture taken eight hours after admission showed much less air in the mediastinum than on admission. There was a steady decrease in respiratory rate, with a return to normal at the end of 48 hours. The pulse rate was normal in 72 hours. There was a slower but a steady decrease in subcutaneous emphysema until it was absent at the time of discharge, on the ninth day.

On the seventh day laryngoscopy and tracheoscopy revealed no abnormality.

The patient was given 300,000 units of crystalline procaine penicillin G (crysticillin®) daily. Her course was afebrile. Routine urine tests and complete blood count gave normal results.

CASE 2.—A 4-year-old white boy was admitted to Los Angeles County Hospital at 7:40 p. m. on Aug. 11, 1951. Several hours prior to admission he fell from his bicycle and received a blow across the front of the neck. Immediately swelling appeared in that region. The swelling steadily increased, spreading to the face, chest, and axillae. There was progressive dyspnea, with shallow respirations. There was marked deformity of the upper chest, axilla, neck, and both sides of the face. In these areas crepitations were easily elicited.

There was a red abraded area anteriorly in the skin overlying the trachea. Neither the trachea nor the larynx could be palpated because of deformity of the overlying soft tissues. The lungs appeared markedly emphysematous, with minimal air sounds and hyperresonance to percussion.

Admission x-ray picture showed some submandibular and anterior cervical emphysema, with associated mediastinal emphysema.

Because of the history of cyanosis and continued progression of the dyspnea, 35 minutes after admission a 4 mm. standard Jackson bronchoscope was passed, low tracheotomy performed, and a No. 3 Jackson tracheotomy tube inserted. At surgery massive subcutaneous emphysema of the neck was encountered, but no great deformity or distortion of the upper airway was observed when the bronchoscope was passed.

The next day the subcutaneous emphysema was evident over the abdomen, in the scrotum, and in both upper eyelids. X-ray at that time showed that the subcutaneous emphysema of the neck was less. However, extensive dissection along both the lateral thoracic and the left abdominal wall had occurred. Mediastinal emphysema was less prominent, but right apical pneumothorax had appeared, with only slight compression of the right upper lung.

The patient was given crystalline procaine penicillin G, 300,000 units daily, streptomycin, 0.25 gm. twice daily, tetanus antitoxin, and gas gangrene antitoxin. Special nurses were present until the tracheotomy tube was removed, on the ninth day.

After 48 hours the subcutaneous emphysema steadily decreased until it was absent, on the 10th day. The temperature rose to 100.2 F. rectally on the second and third day but was otherwise entirely normal. The pulse rate was 124 beats and the respiration rate 28 per minute three hours after surgery. Thereafter, they both steadily decreased to normal in 48 hours after admission.

Indirect laryngeal examination on the sixth day showed some mild edema of the supralaryngeal structures, but the cords and glottis appeared normal. The voice was good throughout the period of observation.

DISCUSSION

The precise location of the upper airway break in both these cases cannot be named. However, there is strong evidence from the location of the skin discoloration and the absence of voice change or late laryngeal difficulties that the injury was in the region which received the blow. Although there is an anatomical distinction, clinically it makes little difference if the break is in the soft tissues just above the cricoid cartilage (laryngeal) or just below (tracheal). The problems encountered will be roughly the same.

Etiology.—While all injuries of the trachea are fairly rare, the particular type under discussion is much less common than the open-wound type. There are two

main reasons why this is so. First, the upper airway is protected to a great extent by the chin and surrounding structures, so that injuries in this region are usually self-inflicted. In most instances cutting or stabbing instruments are used. Second, the airway itself has lateral mobility and may ride with a blow from either side. As a result, the closed type of injury usually is produced by a direct anteroposterior blow which traps the trachea between an applied force in front and the rigid non-yielding vertebral column behind. In this group must also be classed the very few instances of so-called spontaneous rupture of the trachea occurring with coughing,[1] labor,[1] sudden motion of the head,[1] and a blow on the chest.[2]

Anatomical and Pathological Considerations.—The pathological sequelae of tracheal rupture all arise subsequent to the release of air into the peritracheal space and the loose connective tissue lying between the superficial and the deep cervical fascia. From there the air may travel along musculofascial lines upward to the face, superficially to the tissues of the chest, arms, and abdomen, and downward into the mediastinum, pericardium, and pleural spaces. Recognition of the possible avenues of progression and their complications is the prerequisite for good management of the patient in these cases.

The route of extension from the neck downward into the chest compartments has been well demonstrated by Goldberg, Mitchell, and Angrist,[3] who, working on cadavers, inserted air beneath the superficial and deep layers of the cervical fascia with a 20 gauge needle. They then watched this air enter the anterior mediastinum, form emphysematous blebs, and rupture through the mediastinal pleura to produce pneumothorax.

This finding is in conflict with the contentions of Macklin and Macklin,[4] who state that the origin of mediastinal emphysema is almost always from rupture of pulmonary alveoli, with extension first along the perivascular sheaths to the mediastinum and then upward into the neck or laterally to the pleural spaces. They believe that origin of these conditions secondary to laceration of the tracheal mucosa is a rarity. While the two opinions appear to be at variance, they are not mutually exclusive, and probably either route of extension can function, depending on the case at hand. Certainly in the two cases under discussion there does not appear to be much doubt as to the location of the primary opening and the direction of extension.

The amount of air escaping apparently does not depend on the size of the opening but is contingent on the interaction of other less-known factors. Dysart's patient [5] had complete traumatic separation of the trachea with little subcutaneous air leak. Tracheotomy was needed for upper airway obstruction produced by the combined

1. Nach, R. L., and Rothman, M.: Injuries to the Larynx and Trachea, Surg., Gynec. & Obst. **76**:614-622 (May) 1943.

2. Foster, J. H.: Management of Cicatricial Stenosis of the Larynx, Ann. Otol. Rhin. & Laryng. **48**:643-648 (Sept.) 1939.

3. Goldberg, J. D.; Mitchell, N., and Angrist, A.: Mediastinal Emphysema and Pneumothorax Following Tracheotomy for Croup, Ann. Surg. **56**:448-454 (May) 1942.

4. Macklin, C. C., and Macklin, M. T.: Pulmonic Interstitial Emphysema and Its Sequellae: An Anatomical Interpretation, in Essays in Biology in Honor of Herbert M. Evans, London, Ont., Canada, University of Western Ontario Medical School, 1943, pp. 337-368.

5. Dysart, B.: Subcutaneous Rupture of the Trachea from the Larynx by External Trauma with Stenosis and Recovery, Laryngoscope **59**:502-550 (May) 1949.

effect of blood clots, cord paralysis, and discontinuity of the trachea. On the other hand, Goldstein [6] reports the deaths of two newborn infants (seven and nine days old) 40 and 150 minutes after accidental needle puncture of the trachea while attempting a jugular venipuncture. At autopsy an opening 1 mm. in width was found in one trachea filled with a small blood clot. The comparison here may not be too valid in view of the wide difference in age and size of the patients.

Fortunately perforations may seal off spontaneously, as happened in the first case presented. This occurrence is probably helped by a build-up of pressure in the subcutaneous tissue after the entrance of air.

Treatment.—Good management of the patients depends on a cogent understanding of the ways in which death may be produced. The following possible causes must be considered:

A. Respiratory
   1. Upper airway—obstruction or closure at the tracheal level
   2. Lower airway—lack of pulmonary aeration due to pneumothorax

B. Circulatory
   1. Tamponade of the great vessels in the mediastinum
   2. Tamponade of the heart from mediastinal and pericardial air block

With injury there may be produced edema, hemorrhage, cord paralysis, or distortion of the upper airway at the laryngeal or the tracheal level. This pathological condition will be evidenced by dyspnea, stridor, retraction, cough, voice change, and bloody sputum. The treatment is tracheotomy below the site of obstruction.

With rapid progression of subcutaneous emphysema and other findings of air escape a tracheotomy may be performed as a decompression procedure permitting air to escape by changing a "closed" injury into an "open" one. This has the following threefold advantage: 1. The increase in pressure which normally is produced by air passing a partially closed glottis is eradicated. 2. The air stream is diverted below the point of trauma. 3. One may be able to localize the exact site of leak at surgery, evaluate the extent of local damage, and possibly repair it. Usually tracheotomy is sufficient to release the pressure and to prevent any significant increase in the air block. However, other procedures may become necessary and should be kept in mind.

Pneumothorax is to be suspected with unequal movements of the chest, widened intercostal spaces, decreased fremitus, hyperresonance to percussion, a shift of the point of maximum impulse and an area of cardiac dullness to the side opposite the hyperresonance, lowering of the liver, and decrease to absent breath and voice sounds. An x-ray picture is confirmatory. The treatment in the face of progressive signs is needle aspiration of the chest. If the pneumothorax continues to re-form, a needle or catheter may be inserted into the chest and tubing extended to an underwater trap for continued drainage.

Massive mediastinal emphysema with pneumopericardium should be suspected with chest pain and compression, especially over the precordium, distention of the

6. Goldstein, P.: Fatal Interstitial and Mediastinal Emphysema Following Accidental Needle Perforation of the Trachea During Jugular Venipuncture, Am. J. Dis. Child. **78**:375-383 (Sept.) 1949.

neck veins, increasing pulse rate with paradoxical pulse (weaker on inspiration), decreasing systolic and pulse pressure, poorly distinguished point of maximum impulse, large area of cardiac dullness, distant heart sounds, and crunching and clicking sounds heard with the heart beat (Hamman's sign).[7] A chest film, not only in the posteroanterior view but also in the lateral view, is of help by showing the large heart shadow and possibly extravasated air. Sometimes also air may be outlined between the pericardium and the heart.

Careful analysis of the above signs and symptoms will emphasize the fact that these are circulatory complications and the immediate cause of death will be circulatory, not respiratory, in origin.

Mediastinal and pericardial emphysema also may be helped by tracheotomy, which prevents progression of the pathological condition in the chest. Another approach is that of making a wide suprasternal collar incision, separating or, if necessary, dividing the ribbon muscles, then following the peritracheal tissues downward into the superior mediastinum by blunt dissection to decompress that region directly. This procedure may be done separately or combined with tracheotomy.

If pericardial air persists in spite of the above therapy and the symptoms of cardiac tamponade are still severe, pericardicentesis by the left parasternal or costoxiphoid route should be considered.

My first case illustrates the successful conservative management of this problem. Because in a good percentage of cases the perforation will spontaneously close, I feel that the conservative route may be elected if there has been an asymptomatic period of at least several hours. When the conservative course is followed, early or suggestive signs of trouble must be sought; pulse rate, respiration rate, and blood pressure should be checked frequently for gradual but persistent and progressive changes. Setups for emergency tracheotomy and pleural, mediastinal, or pericardial aspiration should always be quickly available. In addition, close observation should be extended by both physicians and special nurses.

Treatment in Other Problems and Complications.—There are several serious problems other than those mentioned for which one must be alerted:

1. Rupture of the Esophagus: A severe blow to the neck may not only damage the trachea but also lacerate or sever the nearby esophagus. If completely severed, the esophagus should be closed in layers by the technique of Scott.[8] Using interrupted chromic absorbable surgical sutures U. S. P., the surgeon should close first the posterior muscular wall, then the posterior mucous membrane, with the knots on the inside of the lumen, then the anterior mucous membrane, with the knots external to the lumen, and lastly the anterior muscular layer. Use of a feeding tube for 7 to 10 days after operation will help relieve tension along the suture line, decrease leakage, insure adequate diet, and prevent total atresia.[9]

2. Total Separation of the Trachea: Because with total separation the two ends tend to retract, surgical reapposition is necessary. This is best performed with the

7. Hamman, L.: Spontaneous Interstitial Emphysema of the Lungs, Tr. A. Am. Physicians **52**:311-319, 1937.

8. Lederer, F. L., and Howard, J.: Wartime Laryngeal Injuries, Arch. Otolaryng. **43**:331-343 (April) 1946.

9. Jackson, C., and Jackson, C. L.: Trauma of the Larynx, Arch. Otolaryng. **38**:413-425 (Nov.) 1943.

use of interrupted chromic absorbable surgical sutures U. S. P. If tension is needed to maintain apposition, it is wise to anchor some sutures around the tracheal cartilages. A tracheotomy below the site of trauma, with diversion of the air stream, is wise because a leak at the suture line in the presence of a primarily closed operative incision will produce all the problems of a second subcutaneous rupture after surgery.

3. Late Stricture or Distortion of the Upper Airway: During the follow-up period one must be on the alert for late changes in the region of trauma. Laryngoscopy and tracheoscopy should be performed before the patient leaves the hospital and at any later date as indicated by the patient's course. If a stricture is found, it may be treated by dilatation with bougies, core molds,[10] or, if necessary, excision of the scar followed by skin graft.[11]

SUMMARY

Two cases of subcutaneous injury of the trachea are reported.

The pathological sequelae of the entrance of free air into the peritracheal tissues are discussed.

Management in this type of case is outlined.

10. Cummings, G. O.: Atresia of the Trachea Following Injury: Dilatation with Core Molds, Ann. Otol. Rhin. & Laryng. **49**:801-804 (Sept.) 1940.

11. Erich, J. B.: Treatment of Extensive Stenosis of the Larynx and Trachea, Arch. Otolaryng. **41**:345-350 (May) 1945.

WATER-SOLUBLE CONTRAST MEDIUM FOR BRONCHOGRAPHY

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WITH THE increased use of segmental and wedge resection in the management of surgical disorders of the lungs and bronchi bronchography has increased in importance and in frequency of application as a diagnostic procedure for the accurate localization of diseased bronchopulmonary tissue. The experiences of the last 25 years with the iodized oils routinely used for bronchography have given rise to several valid objections to these mediums. The objections chiefly center about the slow disappearance of these substances from the lungs, with lesser objections to certain of their physical and chemical characteristics. The persistence of the radiopaque organic iodides frequently obscures or confuses subsequent roentgenograms for long periods. The persistence of the oily vehicle is a definite hazard to the patient, for it has irritative qualities which occasionally lead to chronic lipoid pneumonia or to oil granuloma. The presence of the oil also decreases pulmonary function and increases the frequency and severity of postoperative complications, such as atelectasis and pneumonia. The nonfilling of cavities due to the high viscosity of the mediums, the simulation of organic obstruction by secretions due to the immiscibility of the oils with the aqueous bronchial secretions, and the occurrence of iodism, varying in severity from mild to fatal and due to the instability of the iodide compounds, are further objections to the iodized oils, but of these only the last-mentioned objection is truly important.

CHARACTERISTICS OF WATER-SOLUBLE MEDIUMS

In 1948 and 1949, the Swedish workers Morales and Heiwinkel [1] reported on the successful clinical use of a water-soluble, rapidly disappearing bronchographic contrast medium called "xumbradil viscous B" (Astra) which was free of these

The "xumbradil viscous B" used in this study was supplied by Borje Jalar of Astra Pharmaceutical Products, Inc., Worcester, Mass.

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1. (*a*) Morales, O., and Heiwinkel, H.: A Viscous, Water-Soluble Contrast Preparation: Preliminary Report, Acta radiol. **30**:257-266, 1948. (*b*) Morales, O.: Further Studies with Viscous Umbradil (Umbradil-Viskös): Its Indications and Technical Application, ibid. **32**:317-336, 1949.

objectionable features. ("Xumbradil viscous B" was formerly called "umbradil viscous B.") Soon thereafter somewhat similar preparations were introduced in Switzerland by Fischer [2] as "ioduron B" (Cilag), in Germany by Vieten and associates [3] as "perabrodil M viscous" (Bayer), in France by Delie and Sourdat [4] as "diodone viscous" (Guerbet), and in the United States by Peck and associates [5] as methocel®-diodrast® (methylcellulose, Dow; iodopyracet, Winthrop-Stearns).

Composition and Physical Properties.—All these mediums contain 50 to 60% of the same water-soluble radiopaque stable organic iodide, iodopyracet (the diethanolamine salt of 3,5-diiodo-4-pyridone-N-acetic acid), best known in America by the trade name diodrast.® To increase the viscosity of the iodopyracet solution and thus retard its rate of flow, sufficient water-soluble ether derivative of cellulose is added to make a 1 to 3% concentration of the latter. Now all preparations contain the same cellulose ether, sodium carboxymethylcellulose (hereinafter referred to as CMC) or the sodium salt of the glycolic ether of cellulose. Previously methocel®-diodrast® contained the similar methyl ether, methylcellulose, but because of difficulties in sterilization this has recently been replaced by CMC.

There is a marked difference in the viscosity of the different mediums depending on such factors as the type of CMC (that is, whether of high, medium, or low viscosity, which is determined by the source, methods of purification, and methods and completeness of etherification of cellulose); the concentration of CMC; the presence and concentration of additional substances, such as antibiotics, anesthetic agents, such as lidocaine (xylocaine) hydrochloride in "xumbradil viscous B," fungicides, and wetting agents as are in "ioduron B," and dilution with water, isotonic sodium chloride solution, or iodopyracet solution. Because of these differences in viscosity and therefore in behavior, comparison of the various mediums is difficult because the details of the technique of bronchography, as well as the basic nature and the appearance of the bronchogram, vary considerably from medium to medium. In general, the less the viscosity of the medium, the greater is the rate of

2. (*a*) Fischer, F. K.: Die Darstellung des Bronchialbaumes mit wasserlöslichem Kontrastmittel, Schweiz. med. Wchnschr. **78**:1025-1033 (Oct. 23) 1948. (*b*) Fischer, F. K., and Mülly, K.: Beitrag zur Technik der Bronchographie mit wasserlöslichem Kontrastmittel Joduron-B, ibid. **78**:1033-1035 (Oct. 23) 1948. (*c*) Fischer, F. K.: Technik, Indikationen und Ergebnisse der Bronchographie mit wasserlöshlichem viskosem Kontrastmittell (ioduron B), ibid. **80**:723-734 (July 15) 1950.

3. (*a*) Vieten, H.: Die gezielte Bronchographie mit wasserlöslichem Kontrastmitteln, Fortschr. Geb. Röntgenstrahlen **72**:270-281 (Jan.) 1950. (*b*) Ibers, G.; Vieten, H., and Willmann, K. H.: Bronchographie bei Tuberkulose, ibid. **74**:667-676 (June) 1951. (*c*) Vieten, H.: Erfahrungen bei der Bronchographie mit wasserlöslichen Kontrastmitteln, Röntgen-Blatter **4**:80-109 (March) 1951.

4. Delie, P., and Sourdat, P.: Utilisation des produits iodés hydro-solubles pour la bronchographie; remarques techniques, Presse méd. **58**:1156-1158 (Oct. 18) 1950.

5. (*a*) Peck, M. E.; Neerken, A. J., and Salzman, E.: Clinical and Experimental Studies with a Water-Soluble Agent for Bronchography, abstracted in Surgical Forum: Proceedings of the Forum Sessions Thirty-Sixth Clinical Congress of the American College of Surgeons, Boston, October, 1950, Philadelphia, W. B. Saunders Company, 1951, p. 65; (*b*) Clinical and Experimental Studies in the Use of a Water-Soluble Agent for Bronchography, Surg., Gynec. & Obst. **92**:685-692 (June) 1951. (*c*) Salzman, E.; Peck, M. E., and Neerken, A. J.: Methocel-Diodrast: A Viscous Water-Soluble Contrast Medium for Bronchography; Roentgen and Clinical Results in 23 Cases, Radiology **58**:209-214 (Feb.) 1952.

flow and the greater is the tendency to coat the bronchial walls, rather than to fill the lumina. The scattered and somewhat inconsistent data in the literature [6] indicate that "xumbradil viscous B" has a viscosity approximately one-half that of the iodized oils, that the viscosity of methocel®-diodrast® and of sodium carboxymethylcellulose-diodrast® is slightly greater than that of the iodized oils, and that the viscosity of "ioduron B" and of "perabrodil BR" 60% viscous is three to eight times that of the iodized oils.

Removal from the Bronchopulmonary System.—In spite of differences in physical properties, these mediums are similarly removed from the bronchopulmonary system. The iodide component iodopyracet is rapidly absorbed by the vascular network of the lung and immediately excreted in the urine.[7] Roentgenographic visualization of the kidneys, renal calyces, and urinary bladder 15 to 45 minutes after the intrabronchial administration of these mediums has been demonstrated repeatedly. The rate of iodide absorption is increased with the least viscous mediums, during youth, in the presence of increased pulmonary blood flow and by use of large amounts of topical anesthetic agents. It is decreased, and sometimes markedly so, in diseased segments of the bronchopulmonary system. Alveolar dissemination and beginning absorption are apparent roentgenographically within 5 to 10 minutes, are usually complete in one to two hours, and are always complete in 12 to 18 hours, regardless of the medium used and the status of the lung undergoing bronchography. The Italians [8] use variations in the rate and manner of the bronchial, alveolar, and interstitial or vascular phases of absorption of these mediums as a method of study of the functional status of the bronchopulmonary tree. They liken this "functional bronchography" to excretory urography and cholecystography and claim that it gives information of greater significance than that furnished by both the purely morphologic changes usually sought for and the changes in bronchial dynamics more recently publicized by Di Rienzo.[9]

The problems of the manner and rate of removal of the cellulose derivatives and of the nature, extent, and duration of the damage produced in the lungs by the intrabronchial administration of water-soluble contrast mediums must be considered as not yet completely clarified. Since the cellulose derivatives used are water-soluble, the bulk of any one used is diluted by the bronchial secretions and then removed by the protective mechanisms of the bronchi, namely, peristalsis, ciliary action, and cough.

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6. (*a*) Morales.[1b] (*b*) Fischer.[2c] (*c*) de Fine Licht, E.: Bronchography, with Special Reference to the Use of Ioduron B, Nord. med. **44**:1631-1635 (Oct. 13) 1950. (*d*) Norris, C. M., and Stauffer, H. M.: Aqueous Contrast Media in Bronchography, Ann. Otol. Rhin. & Laryng. **60**:802-816 (Sept.) 1951.

7. Footnotes 1, 2, 3, 4, and 5.

8. Cristiani, M., and Franciosi, A.: Studio della fisiopatolgía bronchiale mediante broncografie esequite con messo di contrasto riassorbible, Radiol. sperimentale **3**:98-115, 1949. Lenzi, M.: Nuovi orientamenti e possibilita' della bronchografia con un mezzio di contrasto riassorbibile: premesse per una "diagnostica broncografica funzionale," Radiologia **6**:21-32 (Jan.-Feb.) 1950. Gambaccini, P.: Considerazioni e vantaggi dall' uso dei composti jodioidrosolubili nella moderna broncografia, Radiol. med. **37**:540-556 (July) 1951.

9. Di Rienzo, S.: Radiologic Exploration of the Bronchus, Springfield, Ill., Charles C Thomas, Publisher, 1949.

The experimental studies of Hellström and Holmgren [10] indicate that any residual CMC is phagocytized by the alveolar and septal phagocytes and completely removed from the lung within two weeks. Temporary storage in the reticuloendothelial cells of the liver and spleen followed by renal excretion was postulated, for this was the sequence of events observed by Hueper [11] after the intravenous administration of CMC. Proof of this hypothesis was furnished by Zollinger [12] by simultaneous intrapulmonary and intraperitoneal injection of "ioduron B." He observed phagocytized CMC in the pulmonary alveoli, alveolar septa, peritoneum, lymph vessels of the omentum and intestinal serosa, and the marginal sinuses of the mesenteric lymph nodes. Nonphagocytized CMC was found in the capillaries of the lung and renal glomeruli and in the collecting tubules of the kidney.

Effect on the Body.—Hellström and Holmgren [10] found that the intrabronchial administration of iodopyracet, "xumbradil viscous B," CMC, and isotonic sodium chloride solution all produced nonspecific inflammatory reactions in the lungs of animals which disappeared completely in about three weeks. The reactions were all of the same type, varying only in severity, with the intensity of the reaction decreasing in the order listed. The CMC had completely disappeared by the end of the second week. Similarly, complete and rapid disappearance of the CMC and nonspecific inflammatory reactions lasting from five days to less than two weeks have been observed in other experimental studies [13] in which the other mediums were used. Absolutely contradictory results were reported by Vischer.[14] He found persistence, for at least four months, of phagocytized and nonphagocytized CMC in the pulmonary alveoli and alveolar septa, with secondary foreign-body reaction and fibrosis in an experimental study with "ioduron B." He found no changes in the spleen, liver, or kidneys which would indicate an excretion of CMC.

The results of examination of human pulmonary tissue obtained at the time of operation or necropsy are as contradictory as the experimental studies. Vischer [14] observed the persistence of CMC with severe inflammation and foreign-body giant cell formation in six surgical specimens removed 6 to 90 days after "ioduron B" bronchography. Zollinger [12] studied 26 specimens removed 6 to 67 days after bronchography with "ioduron B" and 45 control specimens from patients with no prior bronchography. He found that it was possible only to differentiate CMC from mucus with a periodate stain which stained only the mucus, whereas neither the toluidine blue stain used by Hellström and Holmgren nor the thionine tartaric acid

10. Hellström, B., and Holmgren, H.: The Reaction of the Lung on Bronchography with Viscous Umbradil (Umbradil-Viskös B) (Astra), Umbradil, (Astra), and Carboxymethyl Cellulose: An Experimental Investigation on Animals, Acta radiol. **32**:471-485, 1949.

11. Hueper, W. C.: Experimental Studies in Cardiovascular Pathology: Thesaurosis and Atheromatosis Produced in Dogs by Repeated Intravenous Injection of Solutions of Sodium Cellulose Glycollate, Am. J. Path. **21**:1021-1029 (Sept.) 1945.

12. Zollinger, H. U.: Schädigt die Joduron-Bronchographie das Lungenparenchym? Beitrag zur Pathogenese der Schleimgranulome, der xanthomatösen und der interstitiellen Pneumonie bei primären Lungenprozessen, Schweiz. med. Wchnschr. **81**:210-216 (March 3) 1951.

13. (*a*) Morales and Heiwinkel.[1a] (*b*) Morales.[1b] (*c*) Vieten.[3c] (*d*) Zollinger.[12] (*e*) Hentel, W.; Cohen, M. B., and Brandenstein, L. C.: Evaluation of Ioduron B in Bronchography: I. A Preliminary Study on Animal Tissues, Dis. Chest **21**:280-285 (March) 1952. (*f*) Fury, L.: Contribution to Bronchography, Lék. listy **5**:456-464 (Aug. 1) 1950.

14. Vischer, W.: Veränderungen den Lungengewebes nach Bronchographien mit Joduron B, Schweiz. med. Wchnschr. **81**:54-58 (Jan. 21) 1951.

stain used by Vischer [14] could be used for this differentiation because they both stained mucus and CMC in an identical manner. He noted an equally high incidence of granulomas about deposits of epithelial mucus in only the diseased portions of the lungs from both groups of patients. In none of the sections was residual CMC detected. He emphasized that the proof of any claim that CMC is the cause of granulomas is not a positive result with toluidine blue or thionine tartaric acid stain but a negative result with periodate stain. He regarded the inflammatory changes observed by Vischer [14] as being due to retention of mucus, rather than to "ioduron B" bronchography. Vischer [15] replied that he had found these granulomas only in lungs previously subjected to "ioduron B" bronchography, that he had found them in areas of the lung which had not been diseased previously, and that he had found them in the lungs of healthy animals after "ioduron B" bronchography without evidence of retention of mucus.

Vieten [3c] found no damage ascribable to the contrast medium in 42 specimens obtained two days to five months after bronchography with viscous "perabrodil." In answer to a direct inquiry regarding possible injury from "xumbradil viscous B" the research laboratory of its manufacturer replied as follows: [16] "A large undetermined number of surgical and autopsy specimens have been examined for damages from contrast media (Prof. Crafoord's Clinic). It has not been possible to prove that the contrast medium has caused reactions or changes in organs." In 15 specimens removed by resection two weeks to six months after "xumbradil" bronchography we have been unable, with routine histologic technique, to find evidence of damage not ascribable to the preexisting tuberculosis. A detailed study with special staining technique is under way to determine the presence or absence of residual CMC in resected pulmonary tissue.

The problem of systemic injury from the water-soluble contrast medium or its component parts has been studied in man and animals. The long and extensive use of iodopyracet in excretory urography, aortography, and angiocardiography has proved it to be a relatively nontoxic substance. The transient symptoms of headache, nausea, vomiting, and burning paresthesias which sometimes follow its rapid intravenous administration do not occur after its intrabronchial administration, for the rate of entry into the blood stream is much slower under the latter conditions. Severe, even fatal, anaphylactic or immediate allergic reactions, such as are rarely seen after the intravenous administration of iodopyracet, have not yet occurred after the intrabronchial use of these water-soluble contrast mediums, but the remote possibility of their occurrence should be borne in mind. Since the allergy is to the entire molecule, testing for sensitivity to its iodide component is useless. The complete lack of correlation between the results of the various sensitivity tests and these types of severe reactions makes any testing for iodopyracet sensitivity of dubious value. Those with the greatest experience, Vieten and Fischer, and the majority of other workers do not test for sensitivity, whereas a few test for sensitivity to iodopyracet and also to the anesthetic agents.

CMC has been proved to be completely inert and absolutely innocuous when given in large doses by mouth to animals and man; hence, it has a wide use in pharmacology and the food industry. Because it is an inert carbohydrate derivative,

15. Vischer, W., in discussion on Zollinger.[12]

16. Astra Pharmaceutical Products, Inc.: Personal communication to the authors.

it is considered nonantigenic, and no type of allergic reaction or sensitization phenomenon has been noted in man or animal after its oral, intravenous, subcutaneous, intrabronchial, or topical administration. Hueper [11] found that the intravenous administration of comparatively large doses of CMC once, or even on several occasions, produced no gross or histologic evidence of injury, and only after many and frequent infusions of large doses was histologic evidence of injury to blood vessels and storage in the reticuloendothelial system apparent.

In all the numerous experimental studies already cited only Hentel and associates [13e] reported visceral injury. They noted small scattered foci of lymphocytes in the cortical zones of kidneys removed two weeks after "ioduron B" bronchography but not in those removed before or after the two-week period.

The report of Cohen, Hentel, and Brandenstein [17] that "ioduron B" bronchography produced transient impairment of renal and hepatic function in a high percentage of 10 patients is contrary to the far more extensive experiences of all other workers reporting on this subject. An analysis of their data shows them to be inadequate for any type of interpretation, and their conclusions are repudiated, in another publication by Brandenstein,[18] on the basis of additional experience.

From the evidence available it would appear that both components of the water-soluble contrast mediums are probably completely eliminated from the lungs within three weeks and that these mediums produce only transient irritation in the lungs and no injury in other organs.

USE IN BRONCHOGRAPHY

Bronchography with water-soluble contrast mediums has had an extensive trial in Europe since 1948. Vieten [3c] has reported on his personal experiences with 1,000 bronchographies, Fischer [2c] on 500, and numerous other persons in various countries on less extensive series.

The indications and contraindications for bronchography with the water-soluble mediums are essentially the same as with the iodized oil mediums, although bronchography with the former has been much more freely used, particularly in tuberculosis patients,[19] for whom the Europeans consider the use of iodized oil contraindicated.

Various different, but apparently equally successful, techniques are described for performing bronchography with water-soluble mediums. As with other special diagnostic procedures, all require good teamwork, experience, and adequate physical equipment. There can be no doubt that the actual experiences of the examiner play a major role in the choice of anesthesia and also in the choice of methods and of instruments for introduction of the contrast medium into the bronchial system.

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17. Cohen, M. B.; Hentel, W., and Brandenstein, L. C.: Evaluation of Ioduron B in Bronchography: II. A Preliminary Study in Man, Dis. Chest **21**:286-295 (March) 1952.

18. Brandenstein, L. C.: Ioduron B Bronchography: Preliminary Report of Study with Water-Soluble Contrast Medium, Am. J. Roentgenol. **66**:769-772 (Nov.) 1951.

19. (*a*) Footnotes 2 and 3. (*b*) Hoppe, R., and Maassen, W.: Die gezielte Bronchographie mit Métras-Kathetern und einem wasserlöslichem Kontrastmittel bei Lungentuberkulose, Tuberkulosearzt **4**:708-714 (Dec.) 1950; (*c*) Zur gezielten Bronchographie mit wasserlöslichem Kontrastmittel, Deutsche med. Wchnschr. **2**:1730-1731 (Dec. 22) 1950. (*d*) Schultz-Møller, K.: Bronchography with Water-Soluble Contrast Medium, Ugesk. læger **113**:107-116 (Jan. 25) 1951.

All authors emphasize that more nearly complete and more meticulous anesthesia of the tracheobronchial tree is required for these mediums than for the iodized oils. The Europeans all prefer to introduce both a portion of the anesthetic solutions and all the contrast medium into the secondary, or even the tertiary, bronchi through special semirigid catheters. They follow the flow of the contrast medium fluoroscopically and also take serial spot films. The majority of Americans have been reluctant to use bronchial catheterization techniques. They use, instead, semiselective filling by endotracheal catheter and postural manipulation, with or without fluoroscopic observation, and large-film roentgenographic technique. It is probable that the more selective catheter techniques give bronchograms of greater contrast, particularly with the less viscous mediums.

The experiences with these mediums have been most successful—as successful in a research study of bronchial anatomy [20] as in routine clinical usage. They have been used for the localization of foreign bodies,[21] the evaluation of bronchial morphologic changes, and bronchial dynamics, as well as in the study of bronchopulmonary function,[8] as previously mentioned.

The enthusiastic claims of the Europeans that these mediums have solved all or nearly all of the problems of bronchography and have made the iodized oil mediums completely obsolete are tempered by the more conservative, but nonetheless optimistic, evaluation of the American workers.[22]

The objections to these mediums, namely, the problem of increased cough, the rapidity of flow of the least viscous medium, and the incomplete bronchial filling of the more viscous mediums, have been largely overcome by modifications of the mediums or of the techniques employed. The lack of density and contrast of the mediums due to an iodide content approximately one-half that of the iodized oils may be partially compensated for by stereoscopic roentgenograms or spot-film techniques, and the problem becomes less troublesome as one's experience with these mediums increases. There is promise that less irritating water-soluble iodides of greater iodine content may be introduced which will minimize the problems of cough and contrast.

All authors save Huizinga and Métras and their co-workers [23] maintain that the objections are all of minor importance in comparison to the great advantage of complete disappearance of the contrast substance in a few hours and of the vehicle within a few days to two weeks. To date, no one has had comparable experience with several different mediums; hence, the statement of preference for the medium with which the several authors have individually had the greatest experience is not particularly helpful in the selection of the best medium, should such actually exist.

20. Hohn, M., and Vieten, H.: Röntgenologische Studien über die Aufteilung des Bronchialbaumes, Fortschr. Geb. Röntgenstrahlen **73**:669-682 (Dec.) 1950.

21. Willmann, K. H.: Die Bronchographie als Hilfsmittel für die Diagnostik und Lokalisation intrapulmonaler Fremdkörper, Röntgen-Blätter **4**:148-156 (May) 1951.

22. Footnote 5. Norris and Stauffer.[6d] Brandenstein.[18] Atwell, R. J., and Pedersen, R. L.: A Water-Soluble Contrast Medium for Bronchography: Report on Clinical Use, Dis. Chest **18**:535-541 (Dec.) 1950.

23. Huizinga, E., and Smelt, G. J.: Bronchography, Assen, Netherlands, Van Gorcum & Company, Ltd., 1949. Metrás, H.; Charpin, J.; Grégoire, M., and Gaillard, C.: Lipiodol ou produits hydrosolubles pour la bronchographie, J. franç. méd. et chir. thorac. **5**:246-248, 1951.

Complications.—The complications noted after bronchography with these mediums have been due in no case to the mediums themselves but have been those that might occur with any type of contrast medium.

The most frequent complication noted has been elevation in temperature of 1 to 2 degrees F. for a few days following bronchography. Although the incidence of febrile reactions was reported as 53%,[8b] 50%,[19b] and 39%,[5c] respectively, in three series of patients, such reactions usually have been a most infrequent complication. They generally have been ascribed to the endobronchial manipulations, rather than to the introduction of the contrast mediums, for they have also been seen after bronchoscopy and after bronchial catheterization without the introduction of contrast medium.

Death following bronchography with water-soluble contrast mediums has been reported only once.[5c] Details are not available, but the death was regarded as due to idiosyncrasy to the anesthetic agent. Serious, even fatal, anesthetic reactions have previously been reported following bronchial anesthesia with both cocaine and tetracaine (pontocaine®) hydrochloride for bronchoscopy and iodized oil bronchography. It is gratifying that more reactions to the anesthetic agents have not been observed in bronchography with the water-soluble contrast mediums, for the more meticulous anesthesia required with these mediums usually results in the employment of total doses far in excess of the 20 mg. of tetracaine or the 100 mg. of cocaine recommended in the pharmacopoeias. This calculated risk is justifiable only if every safeguard to protect the patient is taken. Therefore, bronchography should be used only in those cases in which other methods of study have failed to give the necessary information and in which it could be expected to have a reasonable chance of furnishing additional information.

Premedication with the opiates had best be avoided, for they are usually unnecessary, and there is evidence suggesting that they potentiate local anesthetic toxicity. On the other hand, premedication with barbiturates should always be used, for they afford marked protection from the toxicity of local anesthetic agents. The use of the smallest amounts of anesthetic agents capable of giving the desired results, the interference with their absorption by the addition of vasoconstrictors, and the avoidance of the ingestion of the agents are further recommended procedures. There may be virtues in the use of combinations, mixtures, or new anesthetic agents, as is sometimes advocated. One should be prepared to treat severe allergic reactions with epinephrine and convulsions or other serious symptoms related to the central nervous system with intravenously administered short-acting barbiturates and sometimes tracheal intubation and artificial respiration.

Dissemination or activation of pulmonary tuberculosis ascribable to bronchography with water-soluble contrast mediums has been reported only in one case,[19b] in spite of rather wide use of these mediums in tuberculous patients and careful search for these complications. In this case the patient with exudative tuberculosis and a large cavity in both apices had an immediate bronchopneumonic spread of the tuberculosis to both lower lobes. The same authors [19b] reported roentgenographic evidence of some increase in the infiltration adjacent to a cavity in another patient unassociated with clinical symptoms. The manufacturers [16] of "xumbradil viscous B" state that Morales, who has had the widest experience with this medium, has never noted spread of tuberculous disease but that two cases had been observed by others in Sweden in which spread followed bronchography with this medium. In

response to a questionnaire sent to numerous workers on their experiences with a total between 2,000 and 2,500 bronchographies with other water-soluble mediums Peck [24] has been informed of only two additional unreported cases of tuberculous spread, and we have been informed of two more. In the one reported and the six unreported cases the spread cannot be definitely ascribed to the contrast mediums themselves, for similar spreads have previously been observed after bronchoscopy and bronchography with other mediums. The Europeans who consider iodized oil bronchography as absolutely contraindicated for patients known or suspected to have tuberculosis use water-soluble mediums freely if the contraindications for iodized oil bronchography outlined by the American workers are applied in the selection of tuberculous patients for bronchography with water-soluble mediums. These contraindications include febrile exudative tuberculosis, recent spread, cavernous disease with expectoration of more than 100 cc. of sputum a day, laryngeal tuberculosis, and recent massive hemoptysis. In addition to the protection afforded by the careful observance of these contraindications, added protection may well be obtained by performing bronchography during the course of combined chemotherapy, but this procedure is not obligatory. The addition of streptomycin and other chemotherapeutic agents to the contrast medium itself has been advocated, but it does not seem probable that the local application of these agents will in any way supplement the effects obtained by their systemic administration.

Dissemination of pyogenic infection has apparently been avoided completely by not doing bronchography during acute inflammatory episodes, by using selective filling of only the involved bronchial segments in the cases of pulmonary abscess, and by using prophylactic chemotherapy. In the three cases of acute pneumonitis observed following bronchography with these mediums the disease was probably of chemical, rather than bacterial, origin. The clinical and roentgenographic manifestations in the two reported cases [25] were similar to those in the case observed by us and are identical with the findings reported previously as occurring after iodized oil bronchography.[26]

Fischer [2c] has noted diminished oxygen saturation with normal or slightly lowered carbon dioxide tension of the arterial blood for as long as 36 hours after bronchography with "ioduron B." He interprets these findings as indicating that pulmonary ventilation is not restricted by filling with contrast medium but becomes irregular. Spirometric studies and blood-gas analysis in a group of silicotics before and after bronchography revealed no other alteration in pulmonary function. Transient dyspnea and even cyanosis have been observed occasionally during the use of the more viscous "ioduron B" due either to tracheal or bilateral bronchial obstruction by medium coughed out of the side being examined or to bronchial obstruction on one side with a contralateral diseased lung possessing an inadequate residual of functioning pulmonary tissue. In the latter patients, usually with extensive bilateral

24. Peck, M. E.: Personal communication to the authors.

25. Ibers, Vieten, and Willmann.[3b] Salzman, Peck, and Neerken.[5c]

26. (*a*) Archibald, E., and Brown, A. L.: Dangers of Introducing Iodized Oil into the Tracheo-Bronchial System, J. A. M. A. **88**:1310-1315 (April 23) 1927. (*b*) Ballon, D. H., and Ballon, H. C.: The Effect of Injection of Lipiodol and the Rate of Its Disappearance in Normal and Diseased Lungs, Canad. M. A. J. **17**:410-416 (April) 1927. (*c*) Bruns, G.: Toxische Pneumonie nach Bronchographie, Zentralbl. allg. Path. **87**:277-280 (June) 1951. (*d*) Fortner, H. C., and Miles, J. S.: Iodized Poppyseed Oil Granuloma: Report of Case, Arch. Path. **49**:447-452 (April) 1950.

bronchiectasis, low-viscosity water-soluble contrast mediums would appear to be the mediums of choice, although iodized oil has been recommended, and the more viscous mediums have been used in the belief that the diminution of respiratory surface produced could be compensated for by the use of oxygen until their reabsorption had taken place.

Iodism has never been noted following bronchography with water-soluble contrast mediums, and the absence of systemic toxicity or local damage following their use has already been discussed.

OUR EXPERIENCES WITH A WATER-SOLUBLE MEDIUM

We have been using "xumbradil viscous B" at Nopeming Sanatorium since January, 1951, for the diagnosis and localization of bronchopulmonary disease that could not be adequately delineated by more routine methods of study. Contraindications were poor general condition, serious renal insufficiency, exudative febrile tuberculosis, hemoptysis in the previous week, and acute respiratory infection. Age itself was no contraindication, the range of ages of our patients being 14 to 70 years. Bronchoscopy was performed in most cases, usually several days or longer before bronchography, although in several cases bronchography was performed immediately after bronchoscopy without additional anesthesia. Tests for sensitivity to the contrast medium and anesthetic agents were not employed routinely.

Techniques.—After trying numerous variations in the technique of anesthesia, of introducing the contrast medium and of taking the roentgenograms, we concluded that the technique to be described was most successful for obtaining diagnostic bronchograms with the unmodified preparation of "xumbradil viscous B."

Since complete cooperation of the patient is most desirable, premedication was minimal, consisting of 100 mg. of pentobarbital (nembutal®) sodium or secobarbital (seconal®) sodium by mouth one and a half hours before the procedure and subcutaneous injections of codeine and atropine three quarters of an hour before.

Topical anesthesia of the pharynx and larynx was obtained by spraying with minimal amounts of a 0.5% solution of tetracaine hydrochloride. Meticulous intrabronchial anesthesia was obtained with a freshly prepared 5% solution of cocaine hydrochloride, first by drop-by-drop transglottic instillation and then by repeated instillation of small amounts through a soft rubber 14 or 16 F. catheter placed into the trachea through the nose or the mouth. As the cocaine was instilled, the patient was positioned as if the contrast medium had been introduced and was encouraged to cough in each position to disseminate the anesthetic agent further. The dose of cocaine was limited to less than 8 cc. of the 5% solution. The patients were encouraged to expectorate and not to swallow the anesthetic agents. No epinephrine or other vasoconstrictor drug was added to any of the anesthetic agents. Consideration was given to the 0.5% lidocaine solution, a local anesthetic agent, present in the "xumbradil" in calculation of the total dose of anesthetic agent used.

For filling the bronchial tree with contrast medium we used the three-position method described by Schmidt.[27] The only modifications of the basic technique were shortening of the time the patient was left in each position and decrease of the angle

27. Schmidt, H. W.: A Method of Obtaining Bilateral Bronchograms, Proc. Staff Meet., Mayo Clin. **23**:71-77 (Feb. 4) 1948; Bronchographic Study of 555 Cases with Special Reference to Bronchiectasis, Ann. Otol. Rhin. & Laryng. **56**:793-803 (Sept.) 1947.

of the head-down positions. For a bronchogram of the left lung the patient is seated on the fluoroscopic table leaning as far backward and toward the left as is conveniently possible without assistance. Care should be taken that the tip of the catheter is high in the trachea; if it is low in the trachea or in a major bronchus, the rapidly flowing medium will flood the alveoli of the lower lobe and inadequately fill the bronchi of the upper lobe. "Xumbradil viscous B," 12 to 15 cc., at room temperature is introduced in about one minute into the catheter with a syringe, and the catheter is flushed out with air. If there is diminished pulmonary volume owing to pneumothorax, pleural effusion, thoracoplasty, prior resection, or small stature, less of the medium should be used to avoid alveolar flooding. The patient is immediately placed on his left side and tipped to a 35 degree angle, head-down position for 50 seconds. The table is righted, the patient is rolled forward into the left anterior oblique position, and again the 35 degree head-down position is maintained for 50 seconds. Upright stereoscopic posteroanterior and single right anterior oblique and left lateral roentgenograms are immediately obtained, using only 5 to 10 kv. more than for the corresponding routine roentgenograms. Fluoroscopic observation is not advisable.

If bilateral bronchography is indicated, both sides are anesthetized initially, and if the patient tolerates, without excessive coughing, the filling of the side suspected of harboring the chief lesion, the second side may be filled after the roentgenograms of the first side have been taken. For a right-side bronchogram a similar three-position method is used: first, the position in which the patient is seated leaning backward and toward the right; second, the right lateral head-down position, and third, the right anterior oblique head-down position. If left-side bronchography has been done at the same sitting, only upright stereoscopic posteroanterior and single left anterior oblique roentgenograms are obtained.

Speed and coordination of efforts are essential throughout the entire procedure, for delays result in disappearance of the medium from the upper lobes and alveolar dissemination in the lower lobes. It should be noted that the time intervals used in the above-described technique were not applicable to another, more viscous, water-soluble contrast medium (methocel®-diodrast®).

Patients Studied and Results.—As of May 1, 1952, we had performed bronchography 79 times on 58 patients with the aid of "xumbradil viscous B." Thirty-eight of the patients were known to have pulmonary tuberculosis; of these, 26 had sputum or gastric washings that were positive for tubercle bacilli, and 12 had pulmonary secretions that were negative for tubercle bacilli at the time of the bronchography. Twenty patients were nontuberculous. Sixty-six unilateral and 13 bilateral bronchograms were made.

In 18 cases two or more "xumbradil" bronchograms were made: in 11 cases to visualize the opposite side, in 6 cases because of previous failure to obtain a satisfactory bronchogram, and in 4 cases to verify apparent pathologic changes.

Sixty-two satisfactory bronchograms were obtained in 79 attempts. There were 17 unsatisfactory bronchograms in 14 cases, but in 7 of these cases satisfactory bronchograms were ultimately obtained: In 4 with "xumbradil," in 2 with iodized oil, and in 1 with methocel®-diodrast.® In the seven remaining failures repeat bronchography with "xumbradil" was unsuccessful in one case, and in this case and in the remaining six cases further bronchography was not attempted. Coughing

due to incomplete anesthesia, nonfilling of portions of the upper lobes, and roentgenographic difficulties arising from the use of spot-film or Bucky-grid techniques were the chief and equally important causes of these failures. Improper position of the tracheal catheter, too much contrast medium, and poor cooperation of the patient were less frequent and largely preventable causes of failure.

The bronchograms obtained when "xumbradil viscous B" was used according to our technique (Figs. 1 to 3) showed much less density and contrast than those usually obtained with the iodized oil mediums and also less than those obtained with "xumbradil" and other water-soluble mediums by bronchial catheterization and spot-film techniques. Although the Swedish workers maintain that satisfactory bronchograms with "xumbradil" can be obtained only by the latter technique, the comparatively light roentgenograms obtained by our technique were of such quality

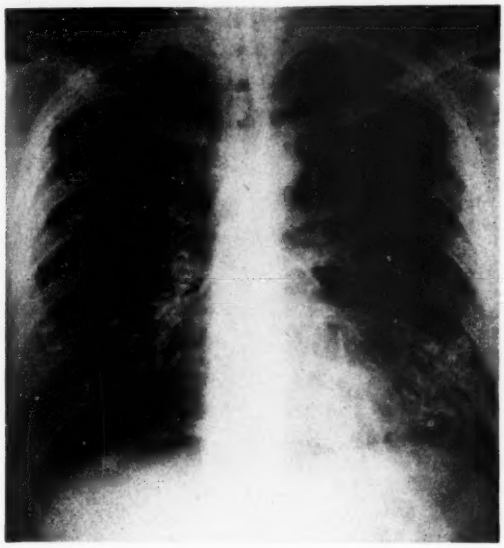

Fig. 1.—Normal bilateral bronchogram showing the clarity on the recently filled right side and the mechanism of clearing by gravity, alveolar dissemination, and vascular absorption on the left side. The bronchogram was made five minutes (left) and one minute (right) after administration of "xumbradil viscous B."

that complete accuracy of diagnosis and of localization was usually possible. When there was any question as to the presence or significance of an abnormality, repeat bronchography was readily carried out within a few days without interference from residual radiopaque material. We have had the opportunity to evaluate the accuracy of our roentgenographic diagnosis in 15 surgical specimens, and in all the preoperative bronchographic diagnosis was confirmed.

No attempt was made to study bronchial dynamics or to evaluate "functional bronchography." Marked alveolar dissemination and beginning vascular absorption of the iodopyracet were routinely observed to take place in the bilateral bronchograms during the five minutes required to fill the second side. Complete disappearance of the radiopaque material usually occurred within one to two hours and always

within 12 hours (Figs. 3 to 5). The rapid disappearance of the contrast medium removed our chief objection to bronchography. As a result, there was a threefold increase in the number of bronchographies performed in the 16-month period covered by this report in comparison with the preceding 16-month period, although the admissions, bronchoscopies, and major thoracic surgical procedures were approximately equal in number for the two periods. "Xumbradil" bronchography proved to be particularly helpful in the preoperative evaluation of the condition of our tuberculous patients both in helping to determine the type and scope of the surgical procedure and in occasionally uncovering contraindications to the contemplated procedure. A significant, but unsuspected, lesion not revealed by any other method of study was sometimes detected by bronchography.

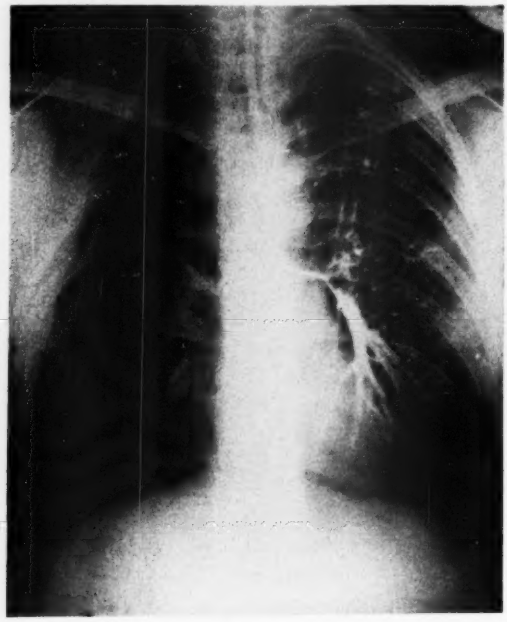

Fig. 2.—Tuberculous bronchiectasis in both divisions of the apicoposterior segment of the left upper lobe without filling of the cavity that obstructs the bronchus of the posterior division.

Complications.—The few complications noted were in no case felt to be due to "xumbradil" itself. Clinically, there was no evidence of toxicity, nor were abnormalities noted in the hemograms or the urine. No evidence of damage to the lungs from the contrast medium was noted in the histologic examination of the 15 specimens obtained by resection two weeks to six months after "xumbradil" bronchography or in the serial follow-up roentgenograms taken in 13 cases for six to nine months, in eight cases for 9 to 12 months, and in five cases for more than one year following "xumbradil" bronchography.

One elderly man became confused on two occasions from much less than the usual dose of topical cocaine, and one young woman had a generalized convulsion during the anesthetization. The convulsion was ascribed to idiosyncrasy, for the

dosage was not excessive, and a similar reaction had been noted in another case after the use of less than 1 cc. of a 0.5% solution of tetracaine in preparation for bronchoscopy. In both cases the convulsion was promptly controlled with a small dose of amobarbital (amytal®) sodium given intravenously.

In none of the 38 tuberculous patients was there hematogenous or bronchial dissemination, and in none of the four in whom evidence of an increase in their tuberculous process was first noted at least three months after bronchography was the bronchography felt to be even contributory. In one of these patients, a boy in whom the clinical and bronchographic picture was typical of epituberculosis with secondary bronchiectasis of the anterior segment of the left upper lobe but from whom tubercle bacilli had never been isolated, enlargement of the right hilar and

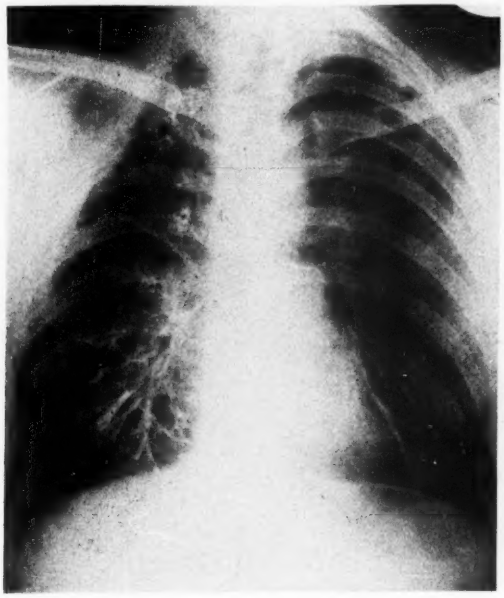

Fig. 3.—Same case as in Figure 2 one week later. Combination of minimal bronchiectasis of the right upper lobe and post-thoracoplasty distortion.

paratracheal nodes developed three months after bronchography of the left lung without roentgenographic evidence of change in the completely left-sided parenchymal lesion. In another patient tuberculous laryngitis developed four months after bronchography without change in the roentgenographic appearance of the cavitary apical lesion. In the third patient six months after bronchography and two months after cessation of a 10-month course of streptomycin and paraaminosalicylic acid therapy slight increase in the roentgenographic shadow cast by a small smoldering noncavitary apical lesion was first observed. In the fourth patient a nodular infiltrate appeared seven months after bronchography and six months after resection of the right middle and lower lobes for tuberculous bronchitis with atelectasis and destruction of the right lower lobe. The nodule appeared in a lobe described as being free of palpable tuberculous lesions at the time of the previous operation

performed during the 10th month of a 19-month course of combined chemotherapy. In the last three patients just mentioned subsequent resection of the disease-containing parts plus additional chemotherapy have resulted in apparent control of their disease.

Although no specific effort was made to perform bronchography during the course of prolonged combined chemotherapy, approximately half the bronchograms were so performed, giving those patients the added protection that such chemotherapy is claimed to afford. From a consideration of the theoretical aspects such planned bronchography might seem preferred. This is not apparent from our data, for no case of spread immediately following bronchography was noted in either group, and in two cases an increase in tuberculous disease taking place many months after bronchography occurred in both groups of patients.

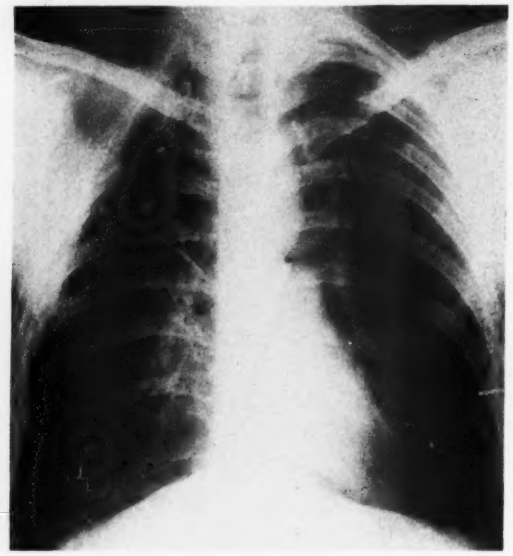

Fig. 4.—Same case as in Figure 3 one hour later. Residual opaque material in the bronchiectatic region and slight diffuse haziness in the lower half of the right pulmonary field due to the presence of iodopyracet in the interstitial tissue of the lung.

One patient experienced an acute pneumonic reaction similar to those described as occurring after bronchography both with iodized oil [26] and with water-soluble contrast medium.[25] She gave a history of recurrent tuberculous cervical adenitis of six years' duration and of moderately advanced right apical cavitary tuberculosis treated by five years of right pneumothorax which had obliterated itself six months previously. While receiving daily combined streptomycin and paraaminosalicylic acid therapy for the cervical adenitis, she had an unsuccessful bronchography in an effort to determine the source of tubercle bacilli isolated by inoculation of a guinea pig with gastric contents after routine roentgenograms of the thorax, planograms, and bronchoscopy had failed to do so. On the morning following bronchography there was complete disappearance of the radiopaque medium, and the roentgenogram was identical with the prebronchographic roentgenogram in that it showed only the

right basal pleuritis. That afternoon she experienced malaise, generalized discomfort, headache, fever, cough, and pain in the right side of the thorax. The symptoms persisted for four days, and the oral temperature which reached a maximum of 102.8 F. by the evening of the first day gradually returned to normal by the fourth day without additional chemotherapy. A roentgenogram taken four days after bronchography showed pneumonitis in the right midpulmonary field (Fig. 6), which completely resolved within the next three days (Fig. 7). The tuberculous adenitis was controlled, no further tubercle bacilli could be isolated by culture or guinea pig inoculation with gastric washings, and no sequelae to the pneumonitis have been observed during the subsequent 11 months.

Another patient, a man aged 47 years, who had quiescent asthma of three years' duration and bilateral fibroid tuberculosis, experienced a delayed type of reaction

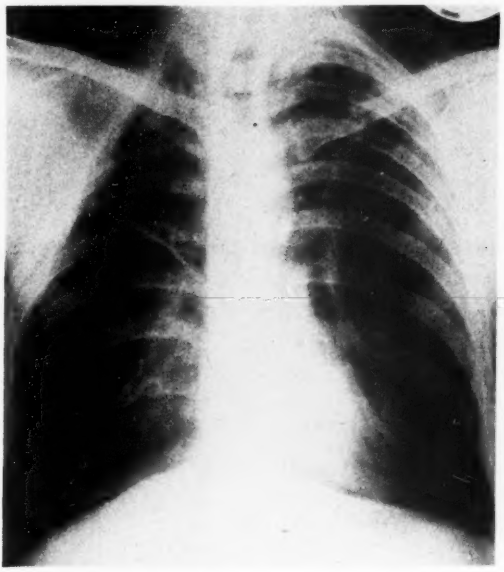

Fig. 5.—Same case as in Figures 3 and 4 two hours and one hour later, respectively. Complete disappearance of all radiopaque medium.

which began two weeks after bronchography. The episode was characterized by onset with cough and increasingly severe asthma, followed in seven days by dyspnea requiring intermittent oxygen therapy, elevation of temperature to 101 or 102 F., and roentgenographic evidence of scattered bilateral small pneumonic infiltrates. The elevation of temperature seemed uninfluenced first by penicillin and then by aureomycin therapy but subsided gradually within a three-week period. The symptoms and pneumonic infiltrates disappeared four weeks after the onset of the episode, and during the subsequent 3½ months no increase in asthma, no return of tubercle bacilli to the gastric washings, and no other sequelae were noted. The etiologic basis for this reaction is most difficult to determine and classify but is considered as probably being related to bronchography, for a similar reaction has

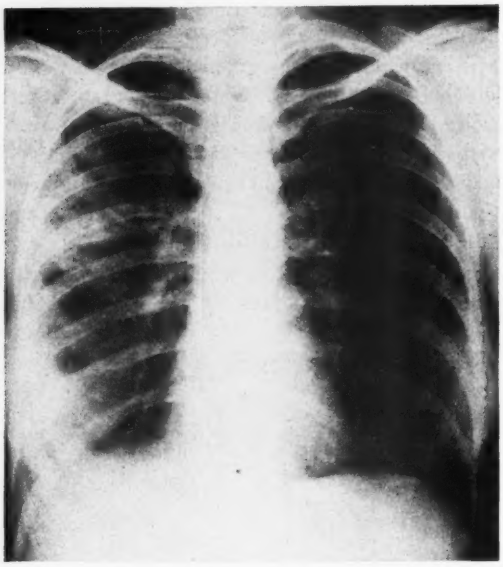

Fig. 6.—Infiltrate in the right midpulmonary field four days after bronchography.

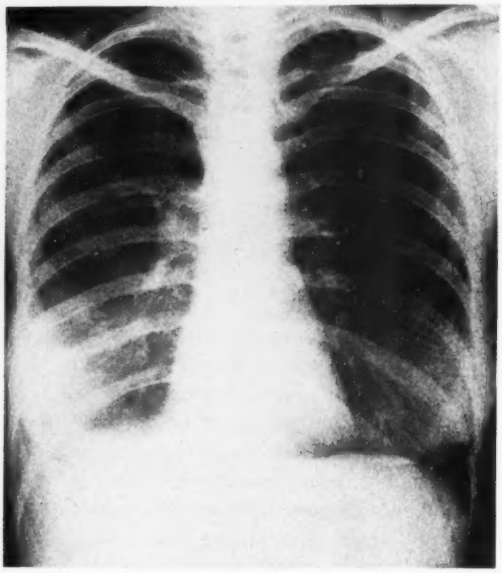

Fig. 7.—Same case as in Figure 6 three days later. Disappearance of pneumonitis. The appearance is identical with that one day before and that one day after bronchography.

been observed following iodized oil bronchography [28] but has never previously been reported as occurring after bronchography with water-soluble contrast mediums. The reaction has occurred chiefly in asthmatics, most commonly associated with persistence of the medium in the lung, and has been ascribed to an allergic factor. In our case a delayed allergic response to the rapidly excreted iodopyracet or to the CMC, which heretofore has always been considered nonantigenic, seems less likely. There is the possibility that the reaction was coincidental, for exactly one year previously the patient had had a three-week episode of severe asthma and fever requiring oxygen therapy.

These were the only two patients in our series who showed any elevation in temperature following bronchography. Iodism or other allergic manifestations were not observed.

Transient dyspnea and slight cyanosis were observed in one case during unilateral bronchography but in none of the 13 cases in which bilateral bronchography was performed.

SUMMARY AND CONCLUSIONS

The definite superiority of solutions of iodopyracet and sodium carboxymethylcellulose as contrast mediums for bronchography has been proved by a rapidly increasing number of experimental and clinical studies. Since they are stable and water-soluble and are completely and rapidly eliminated from the lungs, they appear to possess none of the objectionable features of the iodized oil mediums. They seem to be free of all systemic toxicity and of other than transient local irritative qualities, even with repeated use in the same person. The few complications noted during their use apparently are not due to the mediums themselves but are complications inherent in bronchography. Modifications in the technique of bronchography have largely eliminated the minor objections to these mediums.

Our experience with "xumbradil viscous B" in 79 bronchographies in 58 cases, including 38 in which tuberculosis was present, is in accord with the nearly universally favorable experience of others with this and the other iodopyracet-carboxymethylcellulose mediums.

Dr. H. W. Schmidt assisted in the performance and reporting of this study.

28. Fortner and Miles.[26d] Kooperstein, S. I., and Bass, H. E.: A Pulmonary Reaction Following Intrabronchial Instillation of Lipiodol in Bronchial Asthma, Am. J. Roentgenol. **56**:569-576 (Nov.) 1946. Bass, H. E.: Delayed Pneumonia and Urticaria Following Bronchography, New England J. Med. **240**:505-507 (March 31) 1949. Robertson, P. W., and Morle, K. D. F.: Delayed Pulmonary Complications of Bronchography, Lancet **1**:387 (Feb. 17) 1951.

Clinical Notes, New Instruments and Techniques

NEW OSTEOTOME FOR RHINOPLASTY

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Most operations in rhinoplasty require a thinning of the nose. This is, of course, essential in all cases in which removal of the nasal hump is necessary. To accomplish this properly, both the nasal processes of the maxillary bones and the nasal bones must be brought together in one section. Many methods for performing the lateral osteotomy, with subsequent infracturing of the nasal bones, have been advocated over the years. The proof that an ideal method has not been found is attested by the fact that several methods with variations in techniques are in use today. Each surgeon has periodically alternated among all the present available methods, and the technique being used today does not necessarily dictate the method he will use next month, next week, or even the next day.

The accepted methods available are divided into internal nasal osteotomy and external nasal osteotomy. Internal nasal osteotomies are usually performed by first making an incision several millimeters in length in the pyriform space of the nasal cavity with any type of small sharp blade and by carrying this incision until contact with the lowermost portion of the maxillary process is made. A periosteal elevator is then inserted through this incision and carried along the surface of the bone to make a tunnel along the desired course for the subsequent osteotomy. After this either the Joseph straight or the right angle nasal saw may be used to cut the bone at the desired level. Another method consists of introducing an osteotome through the pyriform incision, and by precise mallet taps on the instrument the osteotomy is easily completed. A combination of the two, use of the nasal saw followed by the osteotome, is often used. I have recently seen J. A. Cinelli use an electrically driven osteotome operating on the trip mechanism of a dental drill to perform the lateral osteotomy. A. P. Seltzer described a circular saw which he devised for the lateral osteotomy. The external nasal approach consists of making a small incision on the skin, taking advantage of its natural folds (lines of Langer), and introducing a 2 mm. osteotome which by gentle mallet blows will cut the nasal process of the maxilla along the desired course.

The methods just described have many limitations. The saws are cumbersome and tiresome to use, and it is often difficult to saw completely the upper portion of the bone near the nasion. The saw is also rather traumatizing to the skin of the nose. The saw blade can easily break in the bone if the overanxious hand of the operator turns or twists the handle as little as 1 or 2 mm. This situation can have disastrous consequences, as a small blade embedded in the hard bone often defies all attempts at removal. The ordinary osteotome used intranasally may cause damage to the frontal bone and occasionally to the frontal sinus if it does not have a guard. If the ordinary guarded osteotome is used, there is danger of breaking the long pointed guard, leaving it embedded in the bone. This is not an infrequent occurrence. Again, it is difficult by this method, particularly for the less experienced operator, to follow accurately the course of the desired cut. There is also danger of the instrument sliding medially or laterally, injuring the nasal skin overlying the osteotomy line or injuring the internal nasal structures, particularly the anterior portions of the turbinates. The external nasal approach for the lateral osteotomy has the disadvantage of an external incision which, psychologically, can be traumatic. Many patients have voiced strong objections and would consider rhinoplasty only on the condition that no skin incision would be made. Scars at the site of incision, even though minute, occur occasionally.

The inherent difficulties and the search for an easier, surer way to perform the lateral nasal osteotomy in rhinoplasties prompted me to incorporate features of several instruments and some

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added new ones into a single new instrument. The instrument is a stainless steel osteotome of the hardest possible steel available (Type 240). It has several features which will be discussed in detail later but which consist essentially of the following: (*a*) a blunt short guard; (*b*) a feeler (guide) on the dorsum of the guard; (*c*) a ledge (collar) along the entire dorsum; (*d*) a blade 6 mm. in width; (*e*) a blade 1 mm. thick for a length of 1½ in. (3.8 cm.), and (*f*) an over-all length of 7 in. (17.8 cm.).

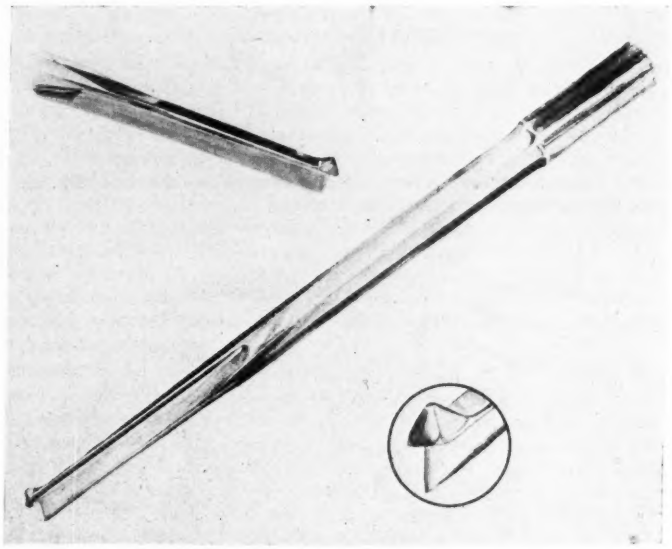

Fig. 1.—The Rish osteotome.

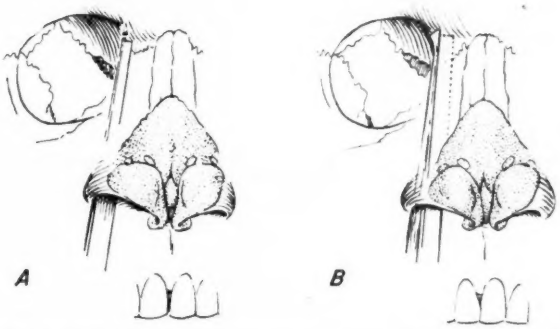

Fig. 2.—*A*, long pointed guard in the conventional guarded chisel can easily break, leaving guard embedded in bone. *B*, blunt short guard cannot possibly injure bone, and the danger of the broken guard embedded in bone is obviated.

(*a*) The blunt short guard is designed to prevent injury to the frontal bone and to prevent it (the guard) from breaking, leaving the fragment embedded in the bone (Fig. 2).

(*b*) The feeler protrudes dorsally and does not have any sharp edges, preventing all damage to the skin. With the fingers of the other hand the exact course of the osteotome can be palpated through the skin, and the treacherous irregular cuts of the ordinary osteotome can thus be avoided (Fig. 3).

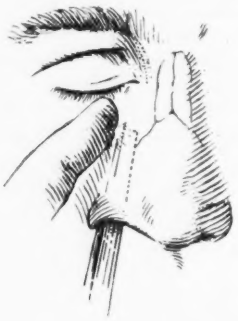

Fig. 3.—The dorsal feeler can easily be palpated through the skin, and thus the osteotome can readily be guided along the desired course.

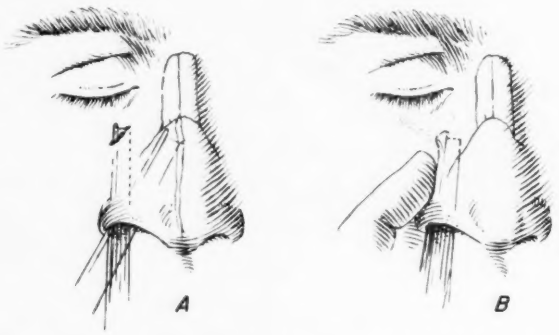

Fig. 4.—*A*, with the conventional osteotome, either guarded or not guarded, there is always the danger of sliding medially, causing damage to intranasal structure, or laterally, piercing the skin. *B*, with pressure exerted medially on the Rish osteotome there is no danger of the instrument sliding laterally or medially because of the ledge (collar) which is in direct contact with bone.

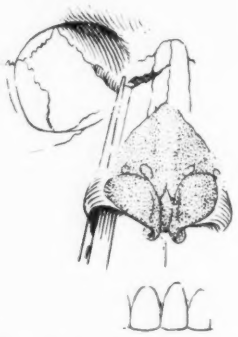

Fig. 5.—Uniform thickness of 1 mm. for a length of 1½ in., as opposed to the conventional osteotome which tapers from the cutting edge backwards, prevents premature separation of the fragments, with undesirable low fracture.

(*c*) There is a ledge (collar) along the entire dorsum. Pressure of the osteotome medially will guide it along the desired course, the ledge obviating the danger of the osteotome sliding medially, causing damage to the intranasal structures, or of the osteotome sliding laterally, injuring the skin (Fig. 4).

(*d*) The blade is 6 mm. wide, which is the maximum width desirable for complete cutting of the maxillary process. At the highest portion, adjacent to the frontal bone, the thickness will not exceed 6 mm. Even in the very occasional case in which the thickness might be exceeded by 0.5 or even 1 mm. (old fractures with a large amount of callus formation), there should be no difficulty in the subsequent fracturing of the bone, as 6 mm. has already been cut through.

(*e*) A blade 1 mm. thick for the entire length of 1½ in. was considered by me to be of paramount importance. Actually, if this blade could be made paper thin, it would be more advantageous. However, there is no steel strong enough which could stand the rigors required of the instrument. With the strongest steel available (Type 240) I found that an osteotome 1 mm. thick would not suffer any damage and that it was thin enough for the operation required. Most osteotomes taper gradually from the cutting edge backwards. This tapering has the disadvantage of spreading the bone fragments as the osteotome advances and very frequently will cause a premature low fracture of the bone, especially since the thickness of the bone increases as one goes cephalad. It was felt that 1½ in. was about the average length of bone that required cutting; therefore the new instrument was made with a uniform thickness of 1 mm. for the length of 1½ in. After this the instrument tapers and becomes, of course, thicker. The uniform thickness without tapering allows the bones to be cut in their entirety without separating the fragments and thus avoids premature low fractures (Fig. 5).

(*f*) Most osteotomes on the market today are approximately 6 in. (15.2 cm.) long. Seven inches in length was considered to be important to facilitate the holding of the instrument comfortably and to prevent the mallet from striking the hand, which is a frequent occurrence.

Actual technique for use of the instrument consists of locating the pyriform space in the nasal cavity. With a scalpel blade a small, 5 mm. incision may be made, or the osteotome itself can be used to cut its own entrance. The osteotome is then engaged by slight pressure with the lowermost portion of the maxillary process. It is not necessary to create a tunnel between the skin and the bone along the desired fracture line, as the guard of the instrument is so designed that it will make its own tunnel, thus avoiding a certain amount of bleeding and trauma to the tissues. The exact course of the instrument is palpated with the finger of the opposite hand resting on the guide. About four precise short blows are required for complete severance of the bone, the entire procedure requiring no more than 10 to 15 seconds for each side. Prior and subsequent to the lateral osteotomy, I, of course, use the accepted method of removing any wedge of bone at the nasion, if indicated, and of separating the nasal bone from the bony septum. After this procedure the bones are fractured in the customary manner by digital pressure.

I believe this instrument has several decided advantages which will help any surgeon doing rhinoplasty, particularly the newer operator in the field, by offering him certain safeguards, as already described, and by enabling him to follow more easily and accurately the desired course of the lateral osteotomy and to achieve more satisfactory and exacting results.

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SECOBARBITAL BURNS

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Some danger seems to exist in leaving secobarbital (seconal®), and presumably other capsuled barbiturate preparations, in contact with mucous membrane. In the past two years I have been twice called between 12:00 and 2:00 A. M. by physicians. In the first instance it was reported that a capsule was lodged in the patient's throat; in the second instance reported, a foreign body, fish bone, was present. Examination of the first patient disclosed a marked swelling of an arytenoid cartilage. There was marked edema on this region. No capsule could be seen. In the second instance it seemed peculiar that the foreign-body sensation of the fish bone should come on so long after the meal. Examination revealed a moderate swelling and edema of the right arytenoid and a pooling of the fluids in the region of the right pyriform sinus. Further questioning accidentally elicited the fact that secobarbital had been taken without water and the pain had come on immediately after this event.

On the basis of this observation there seems little doubt that the capsule caught temporarily and spilled part of its contents in the affected region and that this caused a marked tissue reaction. It is possible that this is due to the alkalinity of this substance, a quality probably shared by other barbiturate preparations.

As the use of barbiturate capsules rectally is a common practice in pediatrics, the question is raised as to whether this may not be harmful in some instances. Of two pediatricians polled on this subject one stated that loose stools were frequent after use, and the other stated that he had noted no such reaction. The rectum is probably much tougher than the lower pharyngeal mucous membrane, and this fact might account for less frequent occurrence of trouble in this region.

These observations indicate the necessity of the patients' washing down these drugs well with water. The attempts at personal pride which indicate this to be unnecessary should be abandoned.

From the Department of Otolaryngology, University of Illinois, College of Medicine.

Progress in Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1951

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PASADENA, CALIF.

IN THE 1951 literature are numerous interesting articles, some of which must have required much time and work in their preparation. Reviewer's notes will appear in brackets.

ANATOMY

Schwartz [1] (Halifax, Nova Scotia) became interested in the influence of heredity on the type of pneumatization in mastoids when he found three persons in one family who had sclerotic mastoids. He x-rayed 25 pairs of identical twins and found their cellular development similar. Seven patients who had had otitis media during infancy were studied, and five had both mastoids well pneumatized, one had diploic mastoids, and one had poor pneumatization. He then x-rayed 13 families, one of them containing 54 members, and recorded the structure types. There were altogether 223 patients x-rayed and studied. He is convinced that heredity is the probable explanation of the different types of bone and the extent of pneumatization present and that Wittmaack's theory is not entirely right. [A very interesting work, his theory should be considered correct as far as it goes. However, many patients have been seen who have a sclerotic mastoid on the side of the discharging ear and a normally pneumatized mastoid on the other side. McMillan, of the Massachusetts General Hospital, published a report of a case of a nurse with pneumatic mastoids, one of which became sclerotic after a mastoid not operated on had drained for several months.]

Altmann [2] (New York) reviews the literature on malformations of the Eustachian tube, middle ear, and appendages. Numerous interesting abnormalities, most of them rare, are mentioned, one of the most important being the variations in the course of the facial nerve. This article covers a great deal of material, listing 272 references in the bibliography. [The article, being a review, does not lend itself well to condensation and should be consulted in the original by those interested in the subject.]

ANTIMICROBIALS

Davidson [3] (Danville, Pa.), in summarizing the treatment of ear, nose, and throat infections, comes to the conclusion that many of the infecting organisms have become much more resistant to penicillin. In 1945, 80% of the cultures were sensi-

1. Schwartz, H. W.: The Influence of Heredity on the Pneumatization of the Temporal Bone, J. Laryng. & Otol. **65**:317-342 (May) 1951.

2. Altmann, F.: Malformations of the Eustachian Tube: The Middle Ear and Its Appendages, A. M. A. Arch. Otolaryng. **54**:241-266 (Sept.) 1951.

3. Davidson, F. W.: The Use of Antibiotics in Otolaryngology, Ann. Otol. Rhin. & Laryng. **60**:207-220 (March) 1951.

tive to 0.2 units per cubic centimeter of penicillin. This percentage gradually decreased until in 1950 none of 50 cultures was sensitive to 0.2 units per cubic centimeter. Many infections now require daily doses of 3,000,000 to 6,000,000 units daily. The addition of 0.5 gm. of streptomycin every six hours increased the efficiency of the treatment for most infections. He also found that many infections would respond to aureomycin or oxytetracycline ("terramycin") if given in doses up to 3 or 4 gm. daily. He has not found an increasing incidence of complications in ear infections, and the percentage of otitis media cases in which simple mastoidectomy is required has decreased from 58.9% to 5.3%. [This same observation about the lack of susceptibility of organisms to penicillin is made by many other writers of this year's articles. Dosages must be increased in most cases to get the effect previously obtained.]

Saunders [4] (Portland, Ore.) reports the results of local applications consisting of one part dihydrostreptomycin and three parts boric acid in both external otitis and in chronic suppurative otitis cases. Between visits at the office he had his patients use alcohol boric acid drops. Thirty-one patients with external otitis were treated, and in 21 the condition was improved. In three patients vesicular dermatitis developed. Of 30 patients with otitis media the same medication improved the condition in 22, and in two vesicular dermatitis developed. [This streptomycin and boric acid powder mixture is very effective. The additional use of the alcohol boric acid drops is not usually necessary and may be painful if the skin is excoriated.]

Evans [5] observed a series of 30 patients admitted to the Massachusetts Eye and Ear Infirmary who had been treated with inadequate doses of antibiotics but without myringotomy. During the same months 48 patients were treated by early myringotomy in the outpatient department. All the 30 patients treated without paracentesis required operation. With two exceptions the dosages had been inadequate. None of the group of 45 treated by early incision required mastoidectomy, and only a few were given chemotherapy; all were cured. He concludes that "myringotomy is an absolute requirement in the early treatment . . . whether or not the use of chemotherapeutic or antibiotic agents are contemplated." He thinks that the "low incidence of surgical mastoiditis in the past few years is to be accounted for by the low incidence of acute suppurative otitis media rather than the result of the routine use of chemotherapeutic antibiotic agents." He also makes the important point that the 30 neglected patients were all under the age of 10 years and that children rarely complain of the impaired hearing which would bring an adult back for further treatment. [These points are all well taken, and one needs to be reminded of the need for watching every patient with otitis media carefully, especially children. But the efficiency of the antimicrobials in the prevention of mastoiditis, if given in proper concentration and for sufficient time, cannot be questioned. And, even though paracentesis is a minor procedure, it can be avoided in most cases if the right antibiotic is given early and in heavy concentrations. One must also take into consideration the type of patient being treated. If the patient is one that is not likely to return for observation, the physician must be more inclined to do paracentesis early.]

4. Saunders, G. C.: Dihydrostreptomycin-Boric Acid Powder in the Treatment of Aural Discharge: A Clinical Report, Laryngoscope **61**:1197-1215 (Dec.) 1951.

5. Evans, M. G.: The Treatment of Acute Suppurative Otitis Media, Ann. Otol. Rhin. & Laryng. **60**:638-647 (Sept.) 1951.

ACUTE OTITIS MEDIA

Rutherford [6] (Oakland, Calif.) emphasizes the importance of careful examination of the ear after apparent cure of ear infections by antibiotics.

> Adequate follow-up demands strong suspicion of residual pathologic process in the ear. . . . The problem of preventing loss of hearing following acute otitis media has been made more complex by the use of penicillin and other antibiotic agents which may apparently cure yet leave dangerous residual disease. . . . In children particularly, loss of hearing may go unnoticed for some time.

She reminds the reader that rupture is caused by ischemic necrosis, a gradual tearing process with pus tunneling through the layers of the tympanic membrane, which leads to greater scarring than a clean incision.

Dr. Goodhill, in discussing the above paper, states that conduction deafness is becoming commoner because of incomplete resolution of infections of the ear inadequately treated. [This need for follow-up observation must be strongly emphasized.]

Hostelley [7] (Philadelphia) writes a very thorough review of the literature on prevention of infections of the ear in swimming. He first covers the problem of examination of water from the public health standpoint and gives rules for the sanitary management of swimming pools. He then quotes at length Taylor's excellent work on rapid chilling of the body in swimmers, with its consequent lowering of resistance to infection. Taylor's conclusions are as follows:

> Chilling of the body surfaces without compensation leads to morbid changes within the body. They are caused (1) by peripheral vasoconstriction with attendant peripheral stasis and anoxemia, (2) by lowered leucocyte response, both total and polymorphonuclear, and (3) by impairment of the phagocytic capabilities of the fixed tissue cells, including that of the nasal mucous membrane. One of the immediate results of these changes is the predisposition to infections of the upper part of the respiratory tract, the paranasal sinuses, the eustachian tubes, and the middle ear.

For keeping water out of the nose Hostelley advises his patients to exhale through the nose while the head is under the water or to wear a nose clip. For his patients who had perforations of the drum he advised cotton plugs of nonabsorbent cotton. The patient was instructed to put four drops of an oily antiseptic in the ear canal first and then to plug the canal with the nonabsorbent cotton. He used a 25% solution of *m*-cresylacetate in chloroazodin (azochloramid®) dissolved in glyceryl triacetate (triacetin). He has found this to be effective not only as a preventive against infection but as an efficient treatment of bacterial and fungous infections. He gives histories of several patients with perforations of the drums who were allowed to swim while using these ear plugs. At the same time treatment of the perforations was frequently successful in healing the perforations.

CHRONIC OTITIS MEDIA

Vicencio [8] (Cebu City, Philippines) devised a very practical and effective method of treatment of chronic otitis media. He added 1 gm. of streptomycin and 200,000

6. Rutherford, M. H.: Proper Use of Antibiotics in Treatment of Acute Otitis Media, California Med. **75**:98-100 (Aug.) 1951.

7. Hostelley, W. V.: Prevention of Otitis in the Swimmer, A. M. A. Arch. Otolaryng. **54**:279-303 (Sept.) 1951.

8. Vicencio, A. B.: Treatment of Chronic Otitis Media with a Mixture of Glycerite of Hydrogen Peroxide, Streptomycin and Penicillin, A. M. A. Arch. Otolaryng. **53**:87-88 (Jan.) 1951.

units of penicillin to 1 ounce of glycerite of peroxide. He treated 30 patients with chronic otitis media with five drops of this mixture four times daily. All ears except one were dry in two weeks, and the remaining one was dry in one month. Two of the patients with mastoid involvement were hospitalized, and streptomycin and penicillin were given parenterally.

CHOLESTEATOMA

Walsh, Covell, and Ogura [9] (St. Louis) report experimental work done to find the underlying factors in the destruction by cholesteatoma. They prefer the term "cholesteatosis." Normal ossicles removed during fenestration operations were immersed in solutions of material removed from patients with cholesteatoma. At the end of three months slight microscopic changes were found which were not present in the controls. They report cases in which destruction has continued after radical mastoidectomy. Therefore, they think that pressure from an expanding mass of accumulating epithelium cannot be the only important factor in destruction of bone. Experiments of implanting of epithelium in the middle ears of rabbits produced no results. They conclude that the exact mechanism is not known. They do advise complete removal of the matrix in cases of cholesteatoma.

In the discussion that follows Williams, Day, and Lindsay agree that the matrix should be removed to prevent recurrences. Lindsay states that when normal skin is kept moist, it desquamates rapidly, and he cites an experiment on monkeys in which a flap pushed into a fenestra produced cholesteatoma that spread right through the labyrinth.

Begley, McDonald, and Williams [10] (Mayo Clinic) review the literature on cholesteatoma and the theories of its formation, giving the arguments for and against each theory. The theories of origin are (1) the primary, or neoplasm, theory; (2) the ingrowth, or immigration, theory, and (3) the metaplasia theory. It is possible for aural cholesteatoma to originate by any of these three methods. The most commonly accepted theory is the ingrowth theory, but the authors think that the metaplasia theory is the only one that can explain all cases. Squamous metaplasia with desquamation of surface layers occurs in many parts of the body in response to irritation, infection, and desiccation. A histological section of cells from an aural cholesteatoma showed prickle cells, establishing the fact that the lining membrane of the middle ear is epidermic in origin. [This article should be read in the original complete paper.]

Ojala and Saxén [11] (Helsinki) examined serial sections of three temporal bones showing pathological changes in Shrapnell's membrane. These were selected from 110 temporal bones. They conclude that a normal Shrapnell's membrane has no lamina propria but does have a differentiated corium which prevents the pneumatization of Prussak's space from the direction of the external auditory canal. This condition promotes the development of cholesteatoma. The prime prerequisite for

9. Walsh, T. E.; Covell, P., and Ogura, J. H.: The Effect of Cholesteatosis on Bone, Tr. Am. Otol. Soc. **39**:125-138, 1951.

10. Begley, J. W., Jr.; McDonald, J. R., and Williams, H. L.: Histology and Histogenesis of Cholesteatoma of the Middle Ear and Mastoid, A. M. A. Arch. Otolaryng. **53**:41-52 (Jan.) 1951.

11. Ojala, L., and Saxén, A.: Pathogenesis of Middle-Ear Cholesteatoma Arising from Shrapnell's Membrane (Attic Cholesteatoma), Acta. oto-laryng., Supp. 100, pp. 33-54, 1951.

the development of cholesteatoma is destruction of the normal relation between epidermis and corium. Indrawing of Shrapnell's membrane does not lead to cholesteatoma, but it may develop without perforation "if there is no dense layer of cicatricial connective tissue corresponding to the lamina propria," since in certain inflammations "the epidermis may project as atypical processes through Shrapnell's membrane."

Volk [12] (Albany, N. Y.) reports a case of cholesteatoma occurring in a woman of 52 without previous history of ear infection. There was a small cyst-like mass protruding from the anterior part of Shrapnell's membrane. Hearing was normal, and x-ray examination showed a normal cellular mastoid. [His conclusion that all cholesteatomas develop as embryonic inclusions is not shared by the majority who have worked over this problem.]

Lewis and Echols [13] (New Orleans) report a case of a man of 28 with neuralgic pain of three years' duration in the right side of the face. He had also progressive complete hearing loss and tinnitus for the same time. The condition was at first diagnosed as tic douloureux, and an alcohol injection gave him relief of pain for 10 months. Operation disclosed a "pearly looking tumor" in the cerebellopontine angle. There was apparent pressure on the 5th, 8th, 9th, 10th, and 11th nerves. After removal of the mass the hearing in the affected ear returned from complete deafness (with masking) to an average of a 20 db. loss. There was no response to the caloric test before operation. No note is made as to whether the labyrinth test was done after operation. The hearing loss returned from complete loss to a 60 db. loss 10 days after the operation and to a 20 db. loss seven months later. [This return of hearing after prolonged compression of the nerve is very interesting.]

Poushter [14] (Detroit) reports an unusual case of cholesteatoma appearing in an infant of 5 months. The infant's first symptom was paralysis of the left side of the face. Spontaneous drainage started in a few days, and examination showed cholesteatomatous material in the pus. There were symptoms of a cold the week before paralysis of the face was noted. A simple mastoidectomy done a week later showed greenish material in the mastoid antrum which the laboratory reported as cholesteatoma. The facial paralysis disappeared in three months, and the drums looked normal. One month later a cold developed in the child, followed in a few days by spontaneous discharge from the opposite ear, and on the next day the facial nerve on that side showed paralysis. Simple mastoidectomy on this ear showed no cholesteatoma. The paralysis cleared up in another month, and the drum appeared normal.

Laub [15] (Columbia, S. C.) reports the case of a 72-year-old man in whom a very large cholesteatoma developed without cholesterol crystals. There had been no complaints except a painless doughy swelling behind the ear for three months, accom-

12. Volk, B. M.: Primary Aural Cholesteatoma, A. M. A. Arch. Otolaryng. **53**:662-667 (June) 1951.

13. Lewis, M. L., Jr., and Echols, D. H.: Pearly Tumor of the Cerebellopontine Angle: Report of a Case with Reversible Deafness, Laryngoscope **61**:1123-1125 (Nov.) 1951.

14. Poushter, D. L.: A Case of Cholesteatoma of the Middle Ear in a 5-Month-Old Infant, Laryngoscope **61**:942-947 (Sept.) 1951.

15. Laub, G. R.: Primary Cholesteatoma of the Mastoid, A. M. A. Arch. Otolaryng. **54**:713-717 (Dec.) 1951.

panied by occasional slight dizziness. The appearance of the drum was normal, and the hearing was apparently normal. The mass, which filled the mastoid, was covered by a thin silky pinkish wall. Microscopic examination showed what seemed to be an epithelial cyst filled with dead epithelial cells without cholesterol crystals. [These last two cases show the extremes of age at which primary cholesteatomas may occur.]

Tamari and Fogo [16] (Chicago) report a case of an unusually large cholesteatoma, weighing 14 gm. It was seen in a man of 42. Because there was no history of previous discharge or other ear symptoms, it was assumed that the cholesteatoma was of the congenital type. Most of the drum membrane was missing. The patient had paralysis of the facial nerve for 1½ years on the side of the cholesteatoma, and the labyrinth was dead. The caloric test produced no dizziness before and after operation. [Several other cases have been reported in which the labyrinthine function was destroyed by infection or pressure erosion and no definite history of previous dizziness could be obtained. Even with no history of previous ear discharge, the finding of a large perforation of the drum would suggest an old middle ear infection and therefore the possibility that the cholesteatoma was of the secondary type.]

MENINGITIS

Nako [17] (Budapest) reports an interesting case of meningitis which developed in a physician of 41, who was an otologist. On the third day of a respiratory infection with symptoms of influenza he had fulness of the ear which he treated by inflation with a catheter. Nako comments that this treatment would not have been done in his department and thinks this may have helped to make the infection worse. On the next day pain in the ear was intense. Myringotomy was done and penicillin treatment started. On the next day there was intense headache and stiffness of the neck, and the spinal fluid showed 27,000 cells. In spite of the fact that the neurologist advised that the meningitis was hematogenous, a mastoidectomy was done the same day. Pneumococci were cultured from the spinal fluid but were not recovered from the mastoid, although there was thin pus present in some cells. The patient recovered after penicillin and sulfonamide therapy. [This case illustrates the danger of active local treatment in respiratory infections. The patient probably recovered in spite of, rather than because of, the mastoidectomy. Early operation in the case of a hematogenous condition is not indicated.]

Elsen, Friedman, and Leshin [18] (Evanston, Ill., and Chicago) report four interesting cases of chronic otitis media with intracranial complications. In all four cases meningitis developed, in two lateral sinus thrombosis developed, and in one, a temporal lobe abscess. One case ended fatally, and the authors criticize their delay in operation (following the advice of the internist who diagnosed the condition as toxic hepatitis). They also criticize their failure to open the lateral sinus.

16. Tamari, M. J., and Fogo, A. E.: True Cholesteatoma of the Temporal Bone, A. M. A. Arch. Otolaryng. **54**:518-523 (Nov.) 1951.

17. Nako, A.: Otitis Media: Initial Meningitis—Recovery, Acta oto-laryng. **39**:56-61 (Feb.) 1951.

18. Elsen, J.; Friedman, E. A., and Leshin, N.: Otogenous Intracranial Complications, A. M. A. Arch. Otolaryng. **54**:409-416 (Oct.) 1951.

Penicillin was apparently not given intrathecally, and they caution that if it is used, it should be given in doses of not more than 10,000 units at a time and in concentrations of not more than 1,000 units per cubic centimeter. They conclude that while the antibiotics are essential to the treatment of complications, the delay of adequate surgery is dangerous, since drugs alone cannot cure an established intracranial complication.

Appleton [19] (Manchester, England) reports four recent cases of otogenic meningitis following chronic infections, with cholesteatosis. Proteus vulgaris was cultured in all four cases. In each case penicillin, 100,000 units, and streptomycin, 100 mg., were given intraspinally until cultures and sensitivity tests were made. In all four cases the Proteus vulgaris was not sensitive to streptomycin. In one case the organism was sensitive to chloramphenicol (chloromycetin®) and not sensitive to either penicillin or streptomycin. Early mastoidectomy was done in each case. Recovery occurred in all four cases.

In two cases the path of invasion was probably through the labyrinth. In both cases there had been labyrinthine symptoms, but since no definite fistula was found in either case, the inner ear was not explored. In one case the path of entrance was the petrous pyramid, and in the fourth case the organism probably entered through the tympanomastoid route. In the fourth case a second operation was needed to drain the petrous pyramid.

LATERAL SINUS THROMBOSIS

Bauer [20] (Liverpool) reports a case of thrombosis of the superior petrosal sinus developing in a man of 40. Both ears had drained for 26 years. For the 10 days before the operation the patient had complained of pain in the left ear and severe toothache in the left lower jaw. At the first operation cholesteatoma was found, but the lateral sinus was not exposed. Bauer points out that he would have saved the patient the second operation if he had recognized that the pain radiating into the lower jaw was caused by irritation of the trigeminal nerve. The second operation disclosed pus in the lateral sinus. There was probably extension into the superior petrosal sinus. The patient recovered without ligature of the jugular vein.

In discussing lateral sinus thrombosis, Elsen, Friedman, and Leshin [18] (previously cited under Meningitis) state that ligation of the lateral sinus should be restricted to those patients who continue to have symptoms of sepsis and bacteremia after adequate local treatment of the thrombosed lateral sinus. This consists of opening the lateral sinus, removing the infected clot until free bleeding is obtained at each end, and packing the ends to control bleeding.

In commenting on their third case, they say as follows:

> The preceding case is a classic example of an attempt to control and cure an otogenous intracranial complication by antibiotics alone. The patient received large doses of penicillin and aureomycin over a period of seven weeks and four weeks, respectively. [This treatment was given at another hospital.] There was no evidence at operation that this had done anything to clear the focus of infection in the mastoid bone or to prevent the spread of infection into the brain.

19. Appleton, J. N.: Otogenic Meningitis: A Report of 4 Recent Cases, J. Laryng. & Otol. **65**:437-441 (June) 1951.

20. Bauer, F.: Thrombophlebitis of the Superior Petrosal Sinus, J. Laryng. & Otol. **65**:199-201 (March) 1951.

BRAIN ABSCESS

Harper [21] (Glasgow) discusses the symptom of aphasia in brain abscess and suggests that it may not be noticed because of the patient's serious condition. He describes in detail the case of a woman of 49 with a temporosphenoidal abscess an inch or more in diameter. The abscess developed after five years or more of chronic otitis media. Aphasia was a pronounced symptom and did not clear up until four weeks after the operation. Aphasia would indicate involvement of the left temporosphenoidal lobe in a right-handed person and of the opposite side if the person is left-handed. The abscess had evidently formed by continuity from an area of decayed bone above the middle ear, and when this bone was removed, an ounce or more of foul pus drained from the abscess through the eroded dura. The literature on the symptoms of sensory and motor aphasia in brain abscess is reviewed thoroughly.

Hughes [22] (Birmingham, England) reports 48 cases of otogenic brain abscesses. Twenty-seven of the lesions were in the temporal lobe and 21 in the cerebellum. There were only eight deaths; three might have been prevented by use of the newer drugs, and three patients were admitted in extremis. Two deaths were ascribed to delayed diagnosis and "appeared to be preventable."

Routine treatment consisted of (1) localization by ventriculography and angiography in about half of the cases; (2) aspiration of the abscess through a burr hole followed by injection of colloidal thorium dioxide (thorotrast®) and sometimes penicillin; (3) removal of the abscess in cases in which further damage to important brain areas would not produce damage. In cases in which it was felt that such removal would create further disability the contracted nodule of fibrous tissue capsule was left in place after repeated aspirations and chemotherapy had reduced its size.

Davis [23] (London) reviews a large number of cases of brain abscess of the cerebellum and points out the paths the infections are most likely to follow.

> Cholesteatomatous abscesses occur in the mastoid antrum and attic. The abscess erodes the roof of the antrum and more frequently extends into the middle fossa, hence a temporo-sphenoidal abscess is more common than a cerebellar abscess. The cholesteatoma exacerbated by acute suppuration erodes into the sigmoid lateral sinus groove, hence a perisinus abscess and thrombosis of the sinus. The largest number of cerebellar abscesses arise from this source.

He mentions that early meningitis confined to the cerebral cortex does not cause pronounced rigidity of the neck, but when basal or posterior meningitis exists, there is always rigidity of the neck. It is a late sign. "There is no doubt whatever that a brain abscess should be tapped at the earliest possible moment after a diagnosis has been made. When the abscess is situated in the posterior fossa any delay results in disaster." Neck rigidity may indicate spontaneous hemorrhage from a vessel of the circle of Willis and was present in 77 cases out of 150. It is also present in most cases of acoustic nerve tumor.

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21. Harper, A. R.: Aphasia as a Symptom of Otogenic Brain Abscess, J. Laryng. & Otol. **65**:89-101 (Feb.) 1951.

22. Hughes, E. B.: Otogenic Brain Abscesses, J. Laryng. & Otol. **65**:207-208 (March) 1951.

23. Davis, E. D. D.: Otogenic Cerebellar Abscess and Some Infections of the Posterior Fossa of the Skull, J. Laryng. & Otol. **65**:646-657 (Sept.) 1951.

Chase [24] (Bethlehem, Pa.) reports the case of a man of 25 in whom labyrinthitis and brain abscess developed. A month after the radical mastoidectomy the brain abscess was opened and the brain tissue allowed to herniate through the wound in the manner described by Dr. Joseph E. J. King. The patient recovered. [With the safety now afforded by antibiotics most neurological surgeons advise complete removal of the infected area without waiting for formation of a capsule around the abscess.]

Chambers and Clark [25] (South Bend, Ind.) report the case of a youth of 16 in whom symptoms of a cerebellar abscess developed nine days after falling and striking his head in a skating accident. He had had a draining ear for six to eight years. There was rapid progress of pressure symptoms, and operation the next day disclosed subdural pus and a tract which led down into an intralobal cerebellar abscess. The culture showed Proteus vulgaris, and the patient recovered after streptomycin, penicillin, and aureomycin therapy. A radical mastoidectomy done later showed a thrombosed lateral sinus and a tract leading into the posterior fossa.

EXTERNAL OTITIS

Collins [26] (Aberdeen, Scotland) discusses the physiology, anatomy, and pathology of external otitis. His article contains excellent reproductions of the gland structure and other histological aspects of the external meatus. He agrees with Senturia that there is no bacteriostatic quality in cerumen. He tried the experiment of filling the ear canals of volunteers with water from one of the local swimming pools and closing the canals with rubber plugs for 24 hours. The same thing was tried using sea water. No infection resulted. Prolonged sweating was produced in volunteers without producing infection. He found that there are no sweat glands in the cartilaginous or bony ear canal.

Vaheri and Savolainen [27] (Helsinki) made cultures in 100 cases of external otitis and found the Micrococcus pyogenes var. aureus commonest in the acute cases and Pseudomonas aeruginosa commonest in the chronic cases. Resistance tests showed wide variations. The Pseudomonas organisms were susceptible only to streptomycin, sulfathiazole, and boric acid. Clinically, the value of the resistance tests was proved.

McLaurin [28] (Louisiana) outlines his general principles of therapy in otitis externa. He stresses thorough cleaning, gentle handling, and the use of simple medications. He still finds 4-aminomethylbenzenesulfonamide hydrochloride (sulfamylon®) one of the most effective medications and used a 1% aqueous solution. He used mild x-ray for very painful cases and advises against the use of most of the antibiotics because of the danger of sensitization.

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24. Chase, W. D.: King Operation for Cerebral Abscess: Report of a Case Complicating Chronic Mastoiditis, with Cholesteatoma and Labyrinthitis, A. M. A. Arch. Otolaryng. **54**:309-311 (Sept.) 1951.

25. Chambers, W. R., and Clark, W.: Bacillus Proteus Abscess of Cerebellar Lobe of Otogenic Origin, A. M. A. Arch. Otolaryng. **54**:179-182 (Aug.) 1951.

26. Collins, E. G.: Certain Observations on the Anatomy, Histo-Pathology and Physiology of the External Auditory Meatus, J. Laryng. & Otol. **65**:14-23 (Jan.) 1951.

27. Vaheri, E., and Savolainen, T.: Bacteriological Examinations and Determinations of Bacterial Resistance in External Otitis, Acta oto-laryng., Supp. 100, pp. 14-19, 1951.

28. McLaurin, J. W.: Principles of Therapy in Otitis Externa, Laryngoscope **61**:66-87 (Jan.) 1951.

If the infections did not respond rapidly, he cultured the secretions and, if necessary, did sensitization tests.

Senturia [29] (St. Louis) did a number of biopsies and presents in his discussion excellent illustrations of the pathological conditions involved in diffuse external otitis. He advises the following as treatment in the acute stage: (1) cleaning by irrigation with hypertonic saline solution; (2) application of hydrous wool fat (lanolin) and boric acid ointment, and (3) administration of antibiotics if the condition is severe. Later, as the condition improves, the following medication is recommended: (1) Burow's solution; (2) metacresylacetate in olive oil (half strength); (3) streptomycin in a polyethylene glycol (carbowax®) vehicle, and (4) vitamin A, 10,000 to 20,000 units per day.

Keller and Meissner [30] (Georgia) report the case of a man whose external otitis was caused by a blastomycosis infection. The middle ear was not infected, but the canal was almost filled with warty pearly granulomatous masses. Treatment consisted of potassium iodide administered by mouth and iodine powder applied to the canal after removal of the granulomatous masses. Healing was complete in 10 weeks. Search of the literature revealed no similar case.

SEROUS OTITIS

Tremble [31] (Montreal) prefers the title "Secretory Effusion of the Tympanum," as it suggests a sterile secretion in the middle ear or the mastoid cells. He states that any obstruction of the Eustachian tube will cause accumulation of serum in the middle ear. He stresses the importance of new growths in the nasopharynx as an important cause that must be kept in mind and searched for carefully. If several inflations with the catheter are not successful, he advises incision of the drum followed by inflation or spot suction.

Robison and Nicholas [32] (Houston, Texas) aspirated fluid in 17 cases of catarrhal otitis media with effusion and injected it into white mice of a strain susceptible to virus disease. Cultures were made into embryonated eggs with similar material. No growth was noted, indicating that the fluid present in the middle ear was sterile. In more than 100 examinations of aspirated fluid no eosinophiles were found. They assume that blockage of the Eustachian tube is caused by hypertrophy of a retropharyngeal lymph node or by lymphedema, with edematous stenosis of the Eustachian tube. Their treatment consists of aspiration of the fluid with a 20 gauge needle and incision of the drum followed by suction or, in case of stubborn conditions, fulguration of the drum to create a semipermanent opening in the drum. They have found radium treatment to be of little value.

Removal of adenoid tissue and of lymphoid tissue along the edges of the Eustachian tube with a special biting forceps is advised. Early recognition of this condition in children is very important. Children are very likely to tolerate a filling of

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29. Senturia, B. H.: Diffuse External Otitis: Its Pathology and Treatment, Tr. Am. Acad. Ophth. **55**:147-159 (Nov.-Dec.) 1950.

30. Keller, A. P., Jr., and Meissner, T. O. W.: Blastomycosis of the External Auditory Canal (Blastomyces Dermatitidis), A. M. A. Arch. Otolaryng. **53**:296-298 (March) 1951.

31. Tremble, G. E.: Secretory Effusion of the Tympanum, Laryngoscope **61**:791-802 (Aug.) 1951.

32. Robison, J. M., and Nicholas, H. O.: Catarrhal Otitis Media with Effusion: Disease of the Retropharyngeal Lymphatic System, South. M. J. **44**:777-789 (Sept.) 1951.

the middle ear with serum without complaint, and it may be recognized only by the slightly orange-colored appearance of the drum. Early and complete removal of adenoid tissue is usually all that is necessary in these cases, but some patients require long-continued care.

MISCELLANEOUS

Schneider and Hegarty[33] (Ann Arbor, Mich.) report two cases of massive extradural hemorrhage occurring as the result of extradural infection eroding vascular channels. Both conditions occurred in young men 21 years of age. One had had recurring otitis media for 15 years, and in the other the condition developed five weeks after a furuncle of the nose had caused swelling and closure of one eye. The first patient died. The autopsy showed massive extracranial hemorrhage, probably from the middle meningeal artery. The second patient was operated upon and a large extradural clot removed from the temporal area. No similar cases were found reported, and the question was raised whether the use of penicillin might have impaired the customary thrombosis of venous channels in the diploë of the skull.

PSYCHOSOMATIC

Braceland,[34] of the department of neurology and psychiatry at the Mayo Clinic, in a talk before the American Laryngological, Rhinological and Otological Society, Inc., reminds the physician that he can help the patient whose chief trouble is emotional disturbance and whose physical condition is secondary. Every disease has a psychic, as well as a somatic, component, and the supposedly sharp distinction between functional and organic condition is extremely nebulous. Emotions must be considered as potential pathological agents. It must be admitted that bacteria "cannot bring about disease unless the organism is prepared for it in a manner physiologic, psychic, or both." Emotional stress limits the organism's ability to resist disease. A physician must ask himself these questions: "What were the original characteristics of the personality of the patients in question? Why did they become ill at the particular time they did? Why did they become ill in the manner in which they did?" These things cannot be seen with the head mirror. [We need to be reminded at times of the importance of the psychosomatic element of our patients.]

33. Schneider, R. C., and Hegarty, W. M.: Extradural Hemorrhage as a Complication of Otological and Rhinological Infections, Ann. Otol. Rhin. & Laryng. **60**:197-206 (March) 1951.

34. Braceland, F. J.: Psychosomatic Medicine and Otolaryngology, Laryngoscope **61**:778-790 (Aug.) 1951.

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708 W. 40th St.
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400 S. Washington St.
Peoria 2, Ill.

Unex Midget Model 95

Unex Midget Model 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 & 66
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)
Mfr., A. N. Brooks Company
684 S. Bonnie Brae St.
Los Angeles 5

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Arthur J. Coombs, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

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The Joint Responsibility of the Internist and the Otolaryngologist in the Removal of Focal Infection. DR. PAUL S. RHOADS (by invitation).

An attempt is made to show that in spite of newer methods of treatment of nose and throat infections, a certain proportion of them progress to a localized chronic infectious focus, which is responsible in part, at least, for such systemic conditions as rheumatic fever, rheumatoid arthritis, iritis, bronchiectasis, and acute and subacute nephritis. Vigorous administration of antibiotics in the acute stage, and even in the subacute or chronic stage, will greatly diminish the number and severity of such foci of infection. Recognition of the allergic nature of many of the chronic pathologic processes in the nose and throat will lead to methods of treatment that may make surgical intervention unnecessary. When such foci exist, however, in spite of a combined antibacterial and antiallergic attack, they often must be removed surgically under the protection of administration of penicillin, aureomycin, or oxytetracycline ("terramycin").

Prevention of these foci by not permitting infections to become chronic and by removing them if they do develop is the joint responsibility of internist and otolaryngologist.

DISCUSSION

DR. WALTER H. THEOBALD: The late Dr. Frank Billings was the pioneer in bacteriology for focal infection. A period of wholesale tonsillectomies and sinus surgery followed. After him came Dr. Ernest Rosenow. Focal infection was so highly specialized at that time that a patient who had torticollis on the right side promptly had a throat culture taken. This was injected into a rabbit, in which subsequently torticollis to the right developed; so Dr. Rosenow named this organism Bacillus torticollis dextra. A second patient had torticollis to the left, and when the procedure was repeated the rabbit manifested torticollis to the left. This organism was named Bacillus torticollis sinistra. Later Dr. Rosenow used organisms that pertained to particular seasons of the year. Vaccines for the fall contained Hemophilus influenzae, streptococci, and pneumococci; when spring came, the entire flora was changed to the organisms most prevalent in that season. The pendulum has now swung back to conservatism.

It is a debatable question whether the internist or the otolaryngologist is the better judge as to a focus located in the nose, sinuses, or epipharynx, or the teeth for that matter. Having had experience with shoulder pain that was the result of a very minute apical tooth abscess, I mention the teeth. Rhinologic examinations require special equipment. I appreciate Dr. Rhoads's daring ability, but I wonder how he can be sure there is an infection in the epipharynx, for example, just because there is an enlarged node in the neck. Biopsy of the node may show a malignant growth in the epipharynx.

Routine temperature recording is an excellent procedure and should be stressed. I find that in our clinics residents have to be told again and again to record the temperature. It is embarrassing to dismiss a patient from the hospital only to find that he has a temperature of 99 or 101 F. on his return home.

Systemic infection as the result of chronic sinus infection is likewise debatable. I fail to find many systemic conditions associated with chronic maxillary sinus disease or ethmoids which are draining or being irrigated.

[Slide] In a critical study of 150 patients with epipharyngeal pathology in which all the evidence was against the epipharynx and all other foci were eliminated, eight were found to

have chronic fever of unknown origin. These patients had been studied thoroughly by an internist, and it was only when the pathology in the epipharynx was discovered, consisting of lymphoid hyperplasia, formation of crypts, or chronically inflnamed furrows, that the diagnosis was established. In one case of asthma we found a pharnygeal bursa as the primary cause. Six additional patients with fever proved to have epipharyngitis; the epipharyngitis was acute and lasted only a few days. One patient with tuberculosis which was primary in the epipharynx also had a tuberculous lesion in the larynx which was previously known.

I point that out because I feel that the otolaryngologist can perhaps better evaluate the presence of pathology in the epipharynx than one can by the finding of a palpable node or nodes in the neck.

DR. SHERMAN L. SHAPIRO: I should like to ask Dr. Rhoads's opininon about an old and discarded method of treatment in the presence of foci of infection in the nose and throat, that is, the use of autogenous vaccines, and what his experience has been in this method of therapy.

DR. O. E. VAN ALYEA: I think that with his recent knowledge of antibiotics and perhaps because of more intensive analysis of the bacteriologic factor than the otolaryngologist ordinarily has time for the internist can offer a great deal of help to the otolaryngologist.

Also, I should like to corroborate Dr. Rhoads's gradual discarding of penicillin mist as a means of therapy so far as nose and throat infections are concerned. Perhaps it would be applicable to chest infections, but I think it is generally felt by otolaryngologists that penicillin in any form locally can better be omitted in treatment of nose and throat conditions.

I wonder whether Dr. Rhoads has encountered a local pharyngeal reaction to aureomycin, a very red throat and edematous palate, such as is occasionally seen. Several such cases every month are observed in persons who have been given aureomycin for several days for treatment of acute pharyngitis. The sore throat does not improve, and when the patient gets to the otolaryngologist there may be a quite pronounced redness and edema.

DR. FLETCHER AUSTIN: Dr. Rhoads brought up the question of allergy. I should like to ask whether he feels that allergy persists for any length of time without the appearance of infection and, at the time of appearance, which is more important in the production of symptoms, the allergy or the infection.

DR. THOMAS C. GALLOWAY (Evanston, Ill.): I have watched the development of Dr. Rhoads's ideas since we were both residents at Cook County Hospital. He has reached his conclusions on a very fine cumulative bacteriologic technic along with clinical observation and has a solid basis for his ideas. I should like to ask him to rate, as to importance, foci of infection. I cannot quite agree than sinuses are so important as foci. It seems to me that the treatment of foci of infection has fallen into disrepute partly because of careless surgical work. If secondary foci are manifest—for example, in heart or joints—it must be obvious that every bit of tissue which harbors infection should be removed. How important are the granular follicles in the pharynx, especially in allergic persons, and the true lingual tonsils?

DR. PAUL S. RHOADS: Perhaps my statements have been a little too sweeping. As to the significance of palpable nodes in the neck, however, I will not withdraw the statements I made. Possibly there is a difference in interpretation. We would be pretty stupid if we could not recognize a hard node that stood out alone as a signal that there might be some malignant condition present. I think that such a node has more significance than a chain of small nodes extending all the way down that are not particularly hard.

I am well aware of our shortcomings in endoscopy of the nasopharynx, and otolaryngologists know more about that than do internists. I draw my conclusions from the symptoms and from the bacteriologic findings in the nose and throat. Small children are more apt to have easily palpable nodes than older children, because they have a larger amount of lymphoid tissue. Clinical experience helps to differentiate a pathologic swelling of nodes from normal lymphatic enlargement. I still think that a chain of nodes in the posterior triangle usually means infection in the nasopharynx, or epipharynx, if that is the proper name.

If one has ever suffered from acute sinusitis due to pneumococci and streptococci, he knows the distressing pain and ache all over the body. Many patients with rheumatoid arthritis have marked infection in the sinuses. Here again we consider the association of a localized lesion with a systemic condition, and I still think that very often such an association exists.

Dr. Galloway asked that I rate the different types of foci according to importance. I do not believe that there is any way of doing that. It depends a great deal on the type of organism found, but I should say that when one sees tonsils with extension of redness to surrounding structures, a great deal of scarring, or small abscesses in the crypts, one is likely to be dealing with important foci. Devitalized teeth are important sources of infection; it has been shown by culture that this is true, even though they do not show rarefaction at the root. But I do not believe that they are foci of infection as often as the tonsils are.

As to lateral lymphoid bands in the nasopharynx, these are the result of drainage from the epipharynx. Dr. Galloway will recall that we made cultures of this lymphoid tissue after it was removed and ground up in sterile broth and often found hemolytic streptococci although surface cultures did not show them. The bands had about the same quantitative bacteriologic content as the tonsils. That type of inflammatory tissue probably can be a real focus of infection. The urinary tract, too, is often such a focus.

Autogenous vaccines given in small doses over a long period, with the idea of desensitization, have a definite field of usefulness in allergic persons, particularly in those with asthma. I doubt, however, that we get a result from stimulating the body to produce large amounts of antibodies, as in immunity against diphtheria. I think an allergic condition can exist in the nose and throat for a long time without infection, in persons who are sensitive to dust or feathers, for instance. Patients who are constantly in contact with pneumococcal and streptococcal infection in this climate are likely to contract infection of the respiratory tract, which will be made worse by the allergic state.

We have seen local reactions to aureomycin. I consider the use of lozenges containing small amounts of antibiotics pernicious. The antibiotic content is not sufficient to kill the microorganisms, but the constant use of these lozenges produces sensitivity reactions. In the case of aureomycin, the growth of yeast is promoted, and that is not good. As I said before, I still believe that there is some field of usefulness for penicillin dust, but it is more limited than was at first believed.

A Study of the Parotid Gland with Reference to the Origin of Some of Its Tumors.
Dr. Elmer A. Friedman.

[This paper was published in full in the September, 1952, issue of the Archives.]

DISCUSSION

Dr. Thomas C. Galloway (Evanston, Ill.): This is a highly technical paper, and it is obvious that Dr. Friedman has been very thorough in his observations. His conclusions seem to agree with those of Ringert, who considered that these tumors arise from a bipolar salivary-type cell with hyaloid secretion. Mucin is closely allied chemically to cartilage. Tumors of the parotid are peculiar. While they do not metastasize early, in my experience they almost always finally undergo malignant degeneration from multiple centers. If they are interfered with, if the capsule is broken, or if they become infected, they change their course completely, grow rapidly, necrose, and produce infection of the surrounding tissue, and the patient usually dies within one or two years. It is important to remember that they are encapsulated tumors. In my experience they do not surround the facial nerve unless they have been tampered with or are late cancers. They must be taken out by the most painstaking dissection, because if some of the capsule is left or if the capsule is ruptured they tend to spread rapidly.

One may speculate that they probably arise from misplaced embryonic rests. About 10 years ago I operated on a 22-year-old nurse, who is now well. Her sister, an identical twin, had a similar tumor operated on two years before and died after the operation. It is interesting that these tumors, even when completely removed, may recur as late as from 9 to 30 years following operation. We are all optimistic about results but cannot be sure until we have had a chance to see our patients very much later.

Dr. Irwin D. Horwitz: We have seen a fair number of these tumors at the tumor clinic of the University of Illinois. We have a slightly different classification: Tumors of the type shown are called mucoepidermoid carcinoma. With the connotation of "carcinoma" added to "mixed tumors," it behooves the general practitioner to treat these tumors in the proper manner.

Dr. Galloway mentioned Ringert's very beautiful work. He explained the myxomatous stroma by bipolar secretion; so if one assumes that the intraductal system becomes blocked, the secretion will break through the acini. If this continues, it will simply swell the loose areolar tissue and form a myxomatous stroma. We call this a fibromyxoepithelial type. This is in contradistinction to the basal epithelioma type, which is subdivided into two types, solid and cystic. The basal epithelioma type is about one-third as common as the myxomatous type.

[Slide] This solid tumor we classify as fibromyxoepithelial type of salivary gland tumor. One theory is that these areas which are sometimes thought to be cartilage are not true chondrocytes and are simply epithelial cells broken off from the acini and caught in the mucoid mesh. They do not act like sarcomas; they act like epitheliomas: They spread by extension, and they do metastasize. Some that we have seen required neck dissection, and we feel that painstaking dissection is necessary. If the tumor surrounds the facial nerve, we feel that we should remove it, whether or not we cut the facial nerve. As Dr. Galloway mentioned, once these are lighted up they become very active. X-rays have no effect; surgery is either a cure or a death warrant. If the capsule is broken during surgery, rapid recurrence will follow. If one is in doubt whether the tumor is benign or malignant, the tumor should be taken out completely and one should forget about the facial nerve. In the clinic we see patients from all over the state whom we cannot help, who should have had more radical surgery in the beginning.

DR. ELMER A. FRIEDMAN: I wish to thank the discussers for bringing in some of the clinical aspects which were not a part of the paper but which, to the practicing otolaryngologist, make it much more interesting.

Intranasal Chondrosarcoma. DR. LAWRENCE J. LAWSON, Evanston, Ill.

A chondroma is a firm, lobulated, grayish, benign tumor. The cells are arranged singly, not in groups. The tumor is relatively nonvascular. It is liable to undergo myxomatous degeneration and often becomes calcified. Growth is slow, and histologically there is evidence of a high degree of differentiation. It is benign but is capable of local invasion and tends to recur.

A chondrosarcoma gives specific evidences of malignancy. These are poorly formed intercellular matrix, increased cellularity, irregular nuclear patterns, and obvious accelerated growth at the margins of the lobules.

Chondroma and chondrosarcoma rarely involve paranasal sinuses and intranasal structures. The intricate anatomy involved usually precludes complete eradication in the sense of eradication with safe margin. The results of irradiation therapy are still disappointing. A new case is presented and another reviewed to illustrate the different pattern of behavior likely to be encountered in chondrosarcoma intranasally at the opposite ends of the life span. Rhinologists faced with the problem will still have to weigh the problem of recurrences following palliative treatment against the problem of functionally and cosmetically undesirable results of attempted complete eradication.

DISCUSSION

DR. JOHN R. LINDSAY: When Dr. Lawson asked me to discuss this paper, I told him that my limited experience scarcely qualified me to do so. The main point I wish to bring up is how to deal with this tumor when it is encountered in a patient in the younger age group. There is no question but that Dr. Lawson's palliative treatment was correct for his patient, since she was 76 years of age.

We are not likely to be helped too much by the pathologist's report, as Dr. Lawson pointed out, because to differentiate between chondroma and chondrosarcoma is not always possible on the basis of the sections. It is true that the age of the patient has to be considered; the rate of spread also must be considered. I think in a young patient one must assume that this could be a sarcomatous type of tumor, or it has the potentialities of one.

It is apparent that we cannot depend on x-ray or radium to eradicate this type of tumor. I think the only hope of getting a satisfactory result in a young patient is by radical exenteration or by removal followed by electrocoagulation in the attempt to destroy any tumor tissue that may have been missed. We must think of this as the basis of adequate treatment. Granted, it will depend on the extent of the tumor when first seen whether such treatment will be possible.

DR. STANTON A. FRIEDBERG: I enjoyed Dr. Lawson's presentation very much and should like to repeat one or two points he mentioned in order to emphasize the relatively slow growth of this tumor and to indicate what can be done in the way of palliation.

[Slide] This patient is 63 years old. Information obtained when we first saw her, in 1948, indicated that the symptoms had apparently existed since 1941 and that a diagnosis of chondrosarcoma had been made as far back as 1944. In that year x-rays showed extensive involvement of all the nasal accessory sinuses. At that time, and again in 1947, she received x-ray therapy, without benefit. You will note the disfiguring enlargement of the nose in 1948. Review of the slides confirmed the diagnosis of chondrosarcoma. There were no clinical evidences of metastases. My colleagues and I felt that some type of operative procedure should be done simply to enable her to breathe and to relieve the deformity of the face.

At operation we left a portion of the nasal tip for prosthetic support, removed a huge tumor of the septum and as much of the massively invading growth from all sinuses as could be visualized. We have used electrocoagulation on several occasions since that time as residual tumor appears. The patient wears a prosthesis and is seen at monthly intervals. She is quite comfortable, there are no metastases, and three and one-half years have elapsed since the operative intervention.

I think this substantiates what Dr. Lawson has said, that these are slow-growing neoplasms and that in the presence of an extensive tumor in an older person a good deal can be done with palliation.

DR. JOSEPH G. SCHOOLMAN: I should like to mention a point with reference to roentgenograms of all sinus tumors, that is, the sclerosing reaction one finds about the frontal sinuses. We speak of this in our group as the Haas sign, because Dr. Haas first called attention to this sign in the literature and has been rather emphatic in calling it to our attention. Another point in x-ray films is the importance of a view of the base of the skull in malignant neoplasms of the sinuses and nasopharynx because of the frequency of extension along the base beyond the scope of surgery. There is no question but that adequate surgery (we do not like the term radical) is necessary in all cases of malignant tumors.

Another point to be kept in mind is the "age" of the patient; not the numerical age, however, because a person of 76 whose parents and grandparents lived to the age of 90 is much younger than one of 60 whose parents and grandparents lived only to their late 50's. That has a good deal to do with determining the life expectancy, and one should consider this factor in terms of definitive treatment.

DR. BRUNO BLUMKLOTZ: I should like to report two cases in which surgery was performed. One was a case of chondrosarcoma of the ear, and a mastoidectomy has been sufficient to keep the patient alive since 1946. The other was a case of chondrosarcoma of the nose and sinuses, and my colleagues and I were able to keep the patient alive for eight and one-half years without radical surgery. He died finally of extension of the tumor into the base of the brain. When we saw him first the tumor had extended from the left ethmoid and antrum through the septum into the right ethmoid and right antrum. There was x-ray evidence of involvement of the left orbital floor. We operated by external and internal surgical approach and the patient was well for three years. He was reoperated on twice and was able to work as a carpenter for seven and one-half years. Finally, we had to remove the left eye and hard palate but were not able to stop the progress of the tumor. To decide on adequate surgery may be difficult when the mutilating effect makes social adjustment of the patient impossible.

DR. THOMAS C. GALLOWAY (Evanston, Ill.): Dr. Soper and I reported a case in which the tumor obliterated the entire sphenoid bone. It was removed with a curette up to the pulsating dura quite satisfactorily by a central "coring" technic, although there was recurrence in four years.

DR. LAWRENCE J. LAWSON (Evanston, Ill.): I wish to thank those who contributed to the discussion. Dr. Lindsay has reemphasized that a different course of treatment may be required, depending on whether the tumor occurs early or late in life. In the young person the tumor is more active and requires more radical treatment.

Dr. Friedberg, in showing the slide of a patient on whom an operation was performed, emphasized that all the evidence and all the indications must be considered in deciding the proper course of therapy.

Dr. Schoolman's comment reminds me that I should stress the desirability of having stereoscopic films to ensure study of the entire extent of the pathology. Some roentgenologists present masked films which leave peripheral regions and the base of the skull unstudied.

Dr. Blumklotz emphasized a point made in the paper, that if surgery is sufficiently radical to destroy the tumor the sacrifice of tissue is such that deglutition and respiration are interfered with, and the cosmetic result makes social adjustment most difficult.

Arthur J. Coombs, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

Dec. 3, 1951

Intralaryngeal Arytenoidectomy for Bilateral Abductor Vocal Cord Paralysis. Dr. William C. Thornell, Cincinnati.

The intralaryngeal approach for the surgical treatment of bilateral abductor vocal cord paralysis is presented. Arytenoidectomy from within the larynx with use of the Lynch suspension apparatus for visualization of the larynx has been found feasible and not particularly difficult for one trained in the use of the suspension apparatus. For those surgeons without such training and equipment the King operation or one of its modifications will and should continue to be the operation of choice.

A few minor modifications have been added to the technique of intralaryngeal arytenoidectomy as presented previously. The removal of the arytenoid cartilage by means of the intralaryngeal approach is carried out with the larynx exposed to direct inspection through the mouth by means of the Lynch suspension apparatus. If the patient is not already wearing a tracheotomy tube, the first step in the procedure is to insert one. This may be done either at the same time as or a few days prior to the arytenoidectomy. After tracheotomy the oropharynx, hypopharynx, and interior of the larynx are anesthetized by topical application of a fresh solution of 5 or 10% cocaine. General anesthesia is then induced by the use of curare and thiopental (pentothal®) sodium administered intravenously, after which the suspension laryngoscope is placed in position. Topical applications of cocaine are next made to the vocal cord and the aryepiglottic fold. An injection of procaine hydrochloride with epinephrine or epinephrine alone may be made into the area to minimize bleeding. An incision 1 cm. in length is made over the superior surface of the arytenoid cartilage, approximately 1 to 2 mm. medial to the cartilages of Santorini and Wrisberg so as to avoid these cartilages in the dissection. The primary incision is carried deep into the tissues down into the arytenoid cartilage. The incision extends anterolaterally into the aryepiglottic fold. By the use of a suitable grasping forceps the superior border of the cartilage is identified. Dissection of the arytenoid cartilage is then carried out. The various muscular attachments are separated from the cartilage in its lateral aspect. The superior portion of the cartilage is then rotated laterally so as to facilitate separation of the muscular attachments on the medial aspect of the cartilage. The cartilage is then removed. A very sharp laryngeal knife, sharp spatula, or scissors is used to carry out the dissection. It is important to avoid tearing or injuring the mucous membrane on the lateral wall of the larynx because scarring in this area may nullify the attempt to increase the glottic opening.

After complete removal of the arytenoid cartilage an electrocautery may be employed into the depths of the wound and lateral to the vocal cord. The tissues within the wound are lightly cauterized on the lateral aspect of the wound only. Lateral fixation of the posterior two-thirds of the cord may be made more certain by the scarring which follows the use of the electrocautery. Many patients have been operated upon in whom an electrocautery has not been used, and the end-results have been successful. At the end of the operation an acrylic obturator may be introduced between the cords to be left in place for a period of three to seven days. The tracheal cannula may be removed after three to six weeks.

With the various improved techniques offered the operating time in performing the intralaryngeal arytenoidectomy has been considerably reduced. This procedure is attended by very little postoperative reaction, and the end-results have been uniformly successsful in Thornell's nine cases, as well as in 61 additional cases reported to him by other surgeons. In four patients of the 70 it was necessary to perform an arytenoidectomy on the opposite side because of an inadequate airway. Excellent results were obtained in three of these patients.

A movie was presented showing the technique of suspension laryngoscopy. Various diagrams were shown demonstrating the technique of the procedure. Recordings of the voice taken before and after surgery were presented.

DISCUSSION

DR. SAMUEL PEARLMAN: I think we all agree that this was a stimulating and an interesting presentation. Two thoughts may be permitted, namely, the effect of bilateral abductor paralysis on the voice and, more important, on the life of the patient. Up to the time of King efforts at cure were sporadic, on occasion based on faulty notions and inadequate from a technical viewpoint. To King, therefore, belongs the credit for initiating successful treatment of a condition which must be faced so long as surgical care for certain thyroid conditions remains the treatment of choice.

It is of interest to dwell upon the two chief approaches toward cure. In one the emphasis was on an approach to the arytenoid cartilage from without, avoiding the interior of the larynx and so rotating the cartilage as to produce the desired result. It must always be a fine point to determine how much airway the patient must be given, for the better the airway, the worse is the voice.

The operations designed to keep the arytenoid cartilage properly rotating outward must remain in the present circumstances difficult, and the performance of them is the prerogative of a few gifted surgeons who have enough material available to become highly qualified. As one reads of the experience of these men, the difficulties become apparent. The attempt to sew the arytenoid in the proper place may tear or break the cartilage, and efforts to expose it properly may, according to most observers, result in a tear into the interior of the larynx, with granuloma formation and a scar which vitiates a possible good result. To escape this difficult project of outwardly rotating the cord and, at the same time, of preserving the arytenoid cartilage, there are those who proceed at once to routine removal of the cartilage. It is claimed that healing and scar contracture will with proper care usually find the cord in the desired position. As a proponent of this approach Dr. Thornell deserves great credit, and his results appear to be as good as any. His approach has the virtue that it is simple if one has the skill necessary to do suspension laryngoscopy.

There are one or two points upon which I hope he will speak. Is it really so that the good result is jeopardized if the interior of the larynx is invaded, or have we feared that contingency too much? Dr. Thornell warns against tearing into the larynx, but his method is an intralaryngeal one. Does he mean that we should avoid undue trauma? The fact remains that intralaryngeal approaches do give good results.

There are members here tonight who have had a large experience similar to that of Dr. Thornell, and I hope that they will share the discussion. As long as there are such cases, and it appears that there will be for a long time, this approach and solution seem to be a long step in the direction of complete success.

DR. PAUL HOLINGER: The problem of bilateral recurrent laryngeal nerve paralysis is one that Dr. Thornell has returned to endoscopic laryngology in a very dramatic way. I should like to congratulate him on his ingenuity and thank him for this presentation. In the past five years my associates and I have seen 72 cases of bilateral abductor nerve paralysis, in 64 of which the disease followed thyroidectomy, and of these we have used the intralaryngeal procedure in 15 cases. Therefore, we add to Dr. Thornell's reported cases an additional series, which further demonstrates the effectiveness of the procedure described. We are convinced that it is practical and gives good results. That statement does not mean that we believe that it replaces other procedures or that we consider other procedures antiquated. Selection of an operative technique is the choice of the surgeon; one may obtain better results with his operation and another with a different procedure; yet, the end-results may be the same in the two series.

One interesting feature of abductor paralysis which we have found important has not been mentioned this evening, that is, the medical management of the patient. We have found that almost every one of our patients has had hypothyroidism and that many of them have had hypoparathyroidism because when the surgeon severed the nerves he had resected too much thyroid tissue and in some patients had also removed the parathyroids. A high cholesterol count and a low blood count are much more significant than a normal or even an elevated metabolism rate. If the patient's thyroid activity is controlled first, his breathing is easier. If it is kept under

control after surgery, he has a better opportunity of maintaining a good airway. It is important to determine the parathyroid activity by obtaining serum calcium levels; not infrequently these patients have respiratory spasms that are corrected by adequate calcium administration. Dr. Keeton and Dr. Ranke have examined our patients carefully in regard to these points, and it is surprising to see how many were myxedematous, in spite of the fact that the basal metabolism rate was normal.

We feel that the use of the plastic obturator is very important, and it is used in our cases. We had the good fortune to have Dr. Thornell help with one of these procedures, and we found, as he suggests, that the plastic mold is of great help.

Dr. Thornell mentioned that the incidence of bilateral paralysis is not related to the skill of the surgeon. I wonder if the use of intratracheal anesthesia may in part be responsible for the increased incidence. The surgeon who does a thyroidectomy without an intratracheal airway is aware that something has happened if one nerve is cut. If an intratracheal tube is used, the patient has no difficulty whatsoever in breathing until the tube is removed. It would seem that the use of intratracheal anesthesia in thyroidectomy adds a certain risk, although it removes what is probably a greater hazard in the majority of cases, namely, respiratory obstruction. Probably with increasing awareness of the tragedy of bilateral paralysis the surgeon may avoid this complication, the incidence of which is increasing so rapidly.

DR. HANS VON LEDEN: I would like to commend Dr. Thornell on his presentation. Several years ago at a meeting of the Academy I saw a preview of this operation in the form of a preserved arytenoid cartilage, which I think he had removed from his first patient. I was impressed with this meticulous dissection and the skill and surgical technique used in the procedure.

In discussing this subject, it is important to evaluate the difference between the extralaryngeal and the intralaryngeal approach by minimizing the imponderables over which the surgeon has no control. As Dr. Pearlman mentioned, and as Dr. Kelly has pointed out with reference to his operation, there are many imponderables in the extralaryngeal approach: the difficult exposure of the arytenoid cartilage by any external approach; the suture material which may break or cut through the cartilage; the formation of granulomata due to mucosal lacerations, and the occasional necrosis of cartilage. Any intralaryngeal operation that eliminates these imponderables over which the surgeon has no control is certainly preferable to the extralaryngeal procedure.

I would like to stress the use of the electric cautery, as mentioned by Dr. Thornell. It is interesting to watch the cord draw aside as the cautery is applied. For me, at any rate, it is a great deal easier to watch the cord moving aside and then to stop at the point at which I think an adequate airway is provided, rather than to rely on someone else looking into the endoscope while I maneuver the suture.

My second point refers to intralaryngeal arytenoidectomy through an external approach. Some months ago I saw a patient with bilateral cricoarytenoid ankylosis, the clinical picture of which is much like the one discussed here. Owing to marked hypertrophy of the false cords in this patient, it was impossible to reach the true cords by the intralaryngeal approach through an endoscope. I therefore decided to perform the arytenoidectomy through a laryngofissure. The thyroid cartilage was split in the midline, the hypertrophied ventricular folds were resected, and one arytenoid was removed without difficulity. Since the arytenoidectomy was performed in accordance with Dr. Thornell's principles, I feel that this case offers further proof that the Thornell operation is a simple, feasible, and desirable procedure.

DR. MAURICE F. SNITMAN: What does Dr. Thornell mean by the term "adequate airway"? Is it that amount which is sufficient to permit removal of the tracheotomy tube only, or does it imply that the patient has enough airway for physical stress? Can he explain the presence of a fairly good voice with sufficient glottic aperture during exertion?

About seven years ago Dr. Galloway advised the removal of the arytenoid cartilage, which procedure I have employed, and in order to obtain a good airway I have also removed some of the thyroarytenoideus muscle. Unfortunately the voice in all cases has been poor, but the patients have ben capable of performing physical tasks.

DR. ROLAND RUSSELL: I should like to mention one point brought up by Dr. Thornell, with which Dr. Holinger has already disagreed. I practice in a small town and see a good many

cases of thyroidectomy. I have seen no case of cord paralysis in the hands of some surgeons who have performed most of the operations. I do not believe that the inferior laryngeal nerve ever passes through the thyroid gland. If the right operation is done, it is not necessary to cut the nerve. I think that one should be charitable to the surgeon and not tell the patient that the inferior laryngeal nerve has been cut.

I agree with the desirability of routine laryngoscopic examination before operation. Several years ago a patient was referred to me because of voice change following thyroidectomy, and I found left cord paralysis. The surgeon said that the operation had consisted of removal of the right lobe of the thyroid, but upon reexamination I still found left cord paralysis.

DR. WILLIAM C. THORNELL: I wish to thank the discussers for their very nice comments. The first question was proposed by Dr. Pearlman. By a tear into the larynx I do not mean just a small opening of a small break in the mucous membrane but maceration of the entire aryepiglottic fold and vocal cord; this could result if one were not familiar with endoscopic surgery. I have had two patients in whom a small tear was produced through the mucous membrane in the region of the vocal process, and the operations on the patients still had successful end-results. Dr. Havens has informed me that on several occasions he has made a small stab incision in the deepest portion of the wound extending into the subglottic area so as to afford better drainage inferiorly. I would like to emphasize that marked tearing and maceration of the posterior aspect of the vocal cord extending into the arytenoid area and interarytenoid space could produce a scar and prevent lateral displacement of the vocal cord.

Dr. Holinger mentioned the charitable remarks made by Havens (with which I agree) regarding the skill of the surgeon doing thyroid surgery. We tried to be fair toward our general surgical colleagues. I have observed abductor vocal cord paralysis as a complication of thyroid surgery performed by some outstanding and skilled surgeons, and when such men encounter that complication, I feel that I should not be one to criticize.

Dr. Snitman asked whether an adequate airway could be obtained with such excellent voice as heard on the recording. In many of my patients close approximation of the anterior third of the vocal cords has developed, with some bowing of the posterior two-thirds, so that on phonation there is tensing of the vocal cord and movement of the cord to the median line, which results from contraction of the cricothyroid muscle which is supplied by the motor branch of the superior laryngeal nerve. This condition produces some approximation of the vocal cords and hence will help in the production of good voice. I have not had the opportunity to observe many patients who have had an external procedure carried out. I have tried the procedure only once and was dissatisfied with the length of time and the difficulty of it. As represented by the drawings, I think that there is further separation anteriorly with the external approach and closer approximation in the patients who had the intralaryngeal approach carried out. Dr. Snitman does not agree with this theory. I have noted in my patients some bowing of the cords, so that on phonation the cords tend to move to the median line, while the glottic opening is large enough to produce an adequate airway.

There is one more point which I believe should be brought up, namely, how can we expect to produce an adequate airway by simply removing the arytenoid cartilage? Simple removal of the space-consuming arytenoid cartilage will produce lateral displacement of the vocal cord. Second the use of an electrocautery will also be a factor in producing further lateral displacement of the vocal cord. Third, freeing of the tension of the interarytenoid muscle may also be a factor so far as this tension alone may hold the cords together in the median position. In the German literature there are reports of cases in which the interarytenoid muscle has been removed, producing some opening of the glottic chink. I have tried severing this muscle previous to intralaryngeal arytenoidectomy but was not too impressed with the lateral displacement of the vocal cords, and so I then proceeded with removal of the cartilage.

Therapeutic Approach to Sudden Deafness. DR. JEROME A. HILGER, Minneapolis.

Sudden deafness, or, to broaden the definition, severe hearing impairment of sudden onset, is a symptom, not a disease entity. The known anatomic, histologic, pathologic, and experimental data implicate vascular and water-balance mechanisms in the production of sudden deafness. Often cited toxic or infectious sources have no substantiating background other than the obvious entity, complicating infectious parotitis. As knowledge develops, other sources may be

identified, but at present sudden deafness must be considered principally under the headings of vascular obliteration, vascular dysfunction, or water imbalance in the cochlear segment of the labyrinth.

Vascular obliteration implies embolus, arteritis, thrombus, or hemorrhage and necessitates careful cardiovascular and blood evaluation in all cases to implicate the few. The majority of cases of sudden deafness result from vascular dysfunction or water imbalance due to endocrine dysfunction. The basal ganglia of the autonomic nervous system together comprise the focal center of autonomic function. Inherent or acquired dysfunction in this central ganglia area can express peripheral imbalance in the cochlea directly through neural routes, indirectly through mediating untoward antigen-antibody reactivity, or humorally through the pituitary axis of the endocrine chain, as dysfunction in the thyroid, adrenals, or gonads.

Representative cases of sudden deafness of these different types are presented, and the therapy is discussed.

DISCUSSION

DR. ROBERT H. EBERT: Dr. Hilger has proposed a most interesting theory and one which deserves careful consideration. I have only one major criticism. The anatomy, physiology, and pathology of small blood vessels are not quite as simple as he has indicated diagrammatically. He has presented the classical concept of vascular physiology. The terminal arteriole is assumed to lead directly into the capillary, and this simple endothelial tube connects with the collecting venule. Spasm of the terminal arteriole, according to this concept, interrupts the blood supply to capillaries and venules completely.

Direct observations by Chambers and Zweifach on living blood vessels have demonstrated that a more complicated anatomic and physiologic arrangement of small vessels exists. According to these authors, the terminal arteriole leads into a metarteriole which has a muscular wall with a single layer of muscle cells which are sparsely scattered at the end of the vessel. The metarteriole leads directly into the collecting venule without traversing any true capillaries. At intervals the metarteriole branches give off precapillaries which lead into the true capillaries. These then drain into collecting venules. At the junction of the precapillary with the metarteriole is a muscular sphincter. Blood may traverse either of these two routes in varying proportions and under different heads of pressure. It may go from arteriole to metarteriole directly into the collecting venule, a circuit in which the filtration pressure is relatively high, or it may go from metarteriole to precapillary to capillary to venule, a system in which the blood pressure falls more rapidly. A third route is often available, that is, a shunt directly from the main arteriole to venule.

In normal vessels there is a rhythmical change in tone and precapillary sphincters, so that more blood is shunted through the metarteriole directly into the venule when precapillary sphincters are constricted and more through precapillaries and true capillaries when these channels are open. This phenomenon is called vasomotion.

The direction of filtration depends upon vasomotion. If a good head of pressure is maintained in the terminal arteriole, enhanced vasomotion tends to increase inward filtration, since the fall of hydrostatic pressure is greater in the true capillaries and the drainage from venules is good. Decreased vasomotion tends to promote outward filtration, since hydrostatic pressure in the capillary bed falls, blood tends to collect in the venule, and increased back-pressure favors an over-all outward filtration. Vasomotion is controlled by both nervous and humoral factors and is a delicately balanced mechanism to regulate fluid and metabolite exchange in the tissues. It is possible, therefore, for certain types of edema to occur as a result of disturbances in the vasomotor mechanism.

If vascular spasm is the cause of sudden deafness, where is it occurring? Is it in the terminal arteriole, metarteriole, or precapillary? These structures do not necessarily constrict at the same time, and the end-results are variable according to which structure is involved. I suspect that prolonged spasm of terminal arterioles sufficient to produce significant anoxia is exceedingly rare. I have seen it occur only in anaphylactic shock. It is possible however, that local change in vasomotion might result in accumulation by anoxia.

I should like to show a motion picture of living vessels in the rabbit ear chamber to illustrate some of these points. [Film.]

I offer these remarks not as destructive criticism of Dr. Hilger's excellent paper but only as a suggestion that the physiologic control of small blood vessels is exceedingly complex and that it is important not to oversimplify the concept of vascular spasm.

DR. JOHN R. LINDSAY: I have greatly enjoyed this lucid exposition of the hypothesis of vascular spasm in relation to inner ear phenomena of various types. One point that should be brought to attention is that although there is a great dearth of information as to what happens to the inner ear in various diseases, gradual progress is being made in the accumulation of fundamental information. For example, we lack information as to the pathologic changes in so-called toxic neuritis; yet, it is common to have severe or profound deafness occur in association with some acute virus infections, such as mumps. Experiments in recent years have shown that the sense organ may be damaged in various ways. We now have information that in streptomycin damage the main and primary injury is to the outer hair cells. The outer hair cells apparently get nourishment directly from the endolymph and not from the vessels. We know that localized damage to the sense organ is caused by acoustic trauma, as well as by severe blows to the head, and that the primary pathologic condition consists in damage to hair cells. More severe trauma causes loss of supporting cells in Corti's organ and degeneration of the peripheral cochlear neurone.

I mention these points to call to your mind that we are accumulating fundamental information and that any hypothesis must take the established information into consideration to be acceptable. Such a hypothesis may be helpful if it gives a rational basis for treatment; yet, we must continue the effort to get the facts. We are indebted, as is otology in general, to Dr. Hilger for his discussion of this phenomenon tonight and over the last few years.

DR. S. L. SHAPIRO: I have considerable sympathy for Dr. Hilger's views, because I have felt for a good many years that many of the internal ear troubles were of vasospastic origin; yet, I think we would all agree that in the absence of direct proof his theory must remain a hypothesis. I have had occasion to use nicotinic acid for inner ear conditions, and, occasionally, by injecting a good stiff dose into the patient in my office, I have been able to abolish temporarily a spontaneous nystagmus. I have never been able to influence tinnitus with it or to obtain any brilliant results in cases of hearing impairment.

Unlike Dr. Hilger, I would hesitate to abolish completely the time-honored concept of neuritis, because we know that such a condition occurs or is considered to occur in other cranial nerves, notably the facial and recurrent laryngeal nerves where paralysis would be difficult to explain on a vasospastic basis.

One place where vascular spasm and the effect of medication upon blood vessels can be observed directly is the eye. A recent article in the *American Journal of Ophthalmology* spoke of a block of the stellate ganglion by the administration of procaine hydrochloride, which produced marked dilatation in a short time of the retinal vascular tree and permitted the passage of an embolus of the central retinal artery, the entire process being observed through the ophthalmoscope. By inference, at least, this experience would prove that stellate ganglion block is one way of obtaining dilatation of cranial end-arteries, and I hope that this method will be tried in cases of sudden deafness in which vasospasm seems to be an important factor.

DR. JEROME A. HILGER: Simplification and unification have a scaffolding purpose in the presentation of this material. In reality the subject of sudden deafness is anything but simple. Obviously there is more ignorance than knowledge of the basic principles involved. One hypothesis which holds that sudden deafness stems from the hypothalamus and operates through humoral and neural channels is supported by many fundamental facts and clinical observations. The gaps in our knowledge are enormous and do not admit of complete correction in the matter of therapy. Yet enough is known of the basic changes in sudden deafness to give a reasonable basis for a varied therapeutic approach and to make unreasonable a "folded-hands," "do-nothing" attitude or a single stereotyped approach to all cases.

The correctness of a hypothesis is in some respects less important than the critical stimulus it may provide to others to deny its accuracy and to formulate real fact.

Abstracts from Current Literature

Ear

USE OF CORTISONE DURING THE POST-FENESTRATION CONVALESCENCE OF OTOSCLEROSIS PATIENTS. H. L. BELL, Eye, Ear, Nose & Throat Month. **31**:605 (Nov.) 1952.

Bell reviews the treatment of some of the complications of the fenestration operation and presents the results of the use of cortisone in a small series of cases. Included among operative accidents in a series of 21 cases of fenestration surgery were three tears of the plastic skin flap, one perforation of the tympanic membrane, one perforation of the endolymphatic labyrinth, and one intralabyrinthine hemorrhage. Among the potential postoperative complications discussed are early serous labyrinthitis, delayed serous labyrinthitis, window closures and emotional disturbances. In this series there were two cases of early serous labyrinthitis and one case of emotional disturbance.

Bell feels that postoperative serous labyrinthitis of the early type, rather than closure of the window, is the most formidable and distressing complication of the fenestration operation leading to irreversible loss of hearing. To reduce labyrinthine irritation, he has utilized cortisone administered intramuscularly, 100 mg. daily for six days, after operation. Though nothing supplants a painstaking and rigidly aseptic technique in reducing the incidence of this condition, it is believed from the observations of the results of the use of cortisone in this small series of cases that cortisone definitely ameliorated labyrinthine irritation, thereby decreasing the incidence of serous labyrinthitis.

JENNES, Waterbury, Conn.

PERCEPTIVE DEAFNESS IN CHILDHOOD. R. SCOTT-MONCRIEFF, Laryngoscope **62**:1081 (Oct.) 1952.

The incidence of severe deafness in children is roughly 1:300 for boys and 1:400 for girls. Of these twice as many have the acquired, postnatal type as the congenital type. A classification table is given for the perceptive deafnesses. Familial deafness is transmitted by the Mendelian pattern. It is a recessive characteristic and results in a developmental anomaly. Rh incompatibility may involve the cerebral cortex and cranial nerves. Maternal rubella affects the cochlear development and is the cause of 20% of infantile deafness. Poliomyelitis also behaves similarly, although the other virus diseases do not. Certain drugs may also affect the development of the cochlea. Of postnatal causes are mentioned infections (meningitis), drugs, and trauma. Diagnosis is discussed, together with methods used to recognize deafness in early childhood. Mental retardation, speech delays, and developmental word deafness are mentioned in the discussion of the differential diagnosis.

HITSCHLER, Philadelphia.

CORRELATION BETWEEN TUBAL STENOSIS AND LESIONS OF THE INNER EAR CAUSED BY SOUND STIMULI. K. TANAKA, J. Oto-Rhino-Laryng. Soc. Japan **55**:596 (Aug.) 1952; **55**:677 (Sept.) 1952.

Innumerable contributions on occupational deafness based on clinical and experimental evidences have appeared since Fosbroke first made his observation, in 1831. The present study is concerned with acoustic trauma in cases of coexisting tubal obstruction in experimental animals.

Preliminary to auditory stimulation, unilateral stenosis was produced by application of diathermy on the right Eustachian tube orifice of guinea pigs. A week later simultaneously the two ears were exposed to continuous noise of equal intensity from a siren with the fundamental frequency of 400 cps, a maximum amplitude of 1200 cps, and a sound intensity of 110 phons. The animals were treated for eight hours daily for 30 consecutive days. Then they were killed. Histologic examination in six animals indicated traumatic lesion in the second turn of the cochlea on both ears. In four animals there was more pronounced change in the cochlea which had tubal stenosis.

In the second experiment one group (A) of animals were exposed to pure tone of 3400 cps at a level of 95 phons for eight hours daily for 30 days. The other group (B) of animals were exposed to a pure tone of 256 cps at a level of 105 phons for eight hours daily on 60 consecutive days. Then both groups of animals were killed and histologically examined. In the first group (A) the lesions in the cochlea were confined to the second turn. In the second group (B) the lesions involved the area from the second to the fourth turn. In all these animals there was observed a greater damage to the inner ear with tubal obstruction.

In the third experiment a group of animals were exposed to blasts of a signal pistol such as is used in athletic races. From 5 to 50 firings were done in a course of one hour above the middle of the animals' heads, and then they were killed. Histologic examination showed that the traumatic lesion was confined to the upper half of the basal turn of the cochlea on both sides, the greater damage being observed on the side which had artificial tubal stenosis.

Tanaka believes that tubal stenosis is a contributing factor to the development of occupational deafness.

HARA, Los Angeles.

Nose

RHINOPLASTIC APHORISMS, REFLECTIONS AND PARADOXES. S. FOMON, I. B. GOLDMAN, H. NEIVERT, and A. SHATTNER, Eye, Ear, Nose & Throat Month. **31**:537 (Oct.) 1952.

In this article the authors list certain generalizations in rhinoplasty justified by the accumulated experience of rhinologists trained in plastic surgery. The pooling of information of rhinoplastic surgeons has resulted in the improvement, simplification, and standardization of the existing methods of rhinoplasty. This article, which touches upon the whole field of rhinoplasty, is extremely concise and should be read in its entirety by all rhinologists interested in plastic nasal surgery. It is written in the form of a series of pithy statements of practical rhinoplastic principles, each followed by a brief explanatory comment.

JENNES, Waterbury, Conn.

THE EFFECT OF CORTOGEN IN RHINOPLASTY. I. B. GOLDMAN, J. FREEMAN, and S. BLOOM, Eye, Ear, Nose & Throat Month. **31**:548 (Oct.) 1952.

The authors report their experiences with the use of cortisone (cortogen®) in rhinoplasty. The problem of postoperative edema and ecchymosis facing the rhinoplastic surgeon has been met by various procedures. Pressure dressings on the eyes for a period of 24 hours, procaine administered intravenously during the course of the operation, and the use of antihistamines and other preparations have limitations, present difficulties, and produce questionable effects. The use of cortisone to prevent secondary swelling and ecchymosis incidental to nasal trauma is based on its effectiveness in altering tissue reaction by hormonal inhibition. As a rule, the degree of reaction depends on that of trauma inflicted during the operation, the variety of local anesthesia used, and the tissue reaction of the patient. The greater the edema, the longer is the period of convalescence and return to nasal normalcy, with diminishing probability of the desired postsurgical result.

The effect of cortisone therapy was demonstrated especially in those patients whose minimal reaction was noted immediately after operation. In the ordinary rhinoplasty in which there has been minimal trauma even slight edema is likely to be followed by ecchymosis, but in this group of patients with slight edema there was frequently no evidence of discoloration. Occasionally a suggestive bluish tint appeared but no real ecchymosis. This result permitted an early return of the patient to his or her vocation. In addition, the psychological impact on the patient and his friends who are influenced by facial appearance after rhinoplasty was most gratifying.

In this series of 70 cases cortisone was administered orally. The preoperative administration varied from 3 to 24 hours prior to the operation. Also, the initial dose of 50 mg. was at times administered at the beginning of the operation and at other times immediately after it. The postoperative dosage consisted of 25 mg. at six-hour intervals for seven doses. The optimum preoperative administration time has yet to be determined. It appears that cortisone should be given from 6 to 12 hours, or even earlier, preceding operations. In a majority of the patients the effect of cortisone was striking, but in others there was little or no influence in the postoperative reaction.

The authors conclude that the interval administration of cortisone before and after rhinoplasty has a distinct inhibitory effect on edema and ecchymosis, with a more rapid return to relative normalcy of the surgical nose. They now use it routinely after plastic surgery of the nose and urge others to evaluate the action of cortisone in rhinoplasty. JENNES, Waterbury, Conn.

NASAL SINUS DRAINAGE: FACTORS WHICH INFLUENCE BERNOULLI ACTION. Z. WILLIAM COLSON, Laryngoscope **62**:1028 (Sept.) 1952.

Colson discusses the various factors concerned in the Bernoulli action which pertain while blowing a congested nose. It is explained why the Bernoulli action is present in some patients and absent in others. Some of the factors are the condensation of the mucus at the sinus ostia, configuration of the air space in the nose at that point, and the amount of pressure produced by the blowing of the nose. All these figure in the efficacy of ridding the sinus of discharge.

HITSCHLER, Philadelphia.

NASAL TUBERCULOSIS AND STREPTOMYCIN. FRANCO CORDARO, Rev. otorrinolaring. **12**:21 (April) 1952.

The author presents a study of a series of four patients whose ages ranged from 13 to 48 years who had definite tuberculosis of the nose verified by histologic study of the tissue. Pulmonary tuberculosis was not found in any of the four patients. Apparently the lesions of the nose were of a primary nature. Dihydrostreptomycin was used in this series of patients; the dosage was 1 gm. daily for a period of seven to eight weeks. The lesions disappeared under this treatment, and the resulting scars had both a gross and a histologic appearance of normal scar tissue. Systemic reactions to the drug were minimal, and the author advocates its use in preference to the mutilating agents, such as the caustics, and to electrocoagulation.

PERSKY, Philadelphia.

Miscellaneous

EPIDERMOID ARISING FROM THE PETROUS PORTION OF THE TEMPORAL BONE. E. D. FISHER and P. J. VOGEL, Bull. Los Angeles Neurol. Soc. **16**:357 (Dec.) 1951.

Fisher and Vogel report the findings in the case of a woman aged 28 who presented the symptoms and signs of a lesion involving the middle fossa on the right side, in addition to those suggestive of a cerebellopontine-angle tumor. Roentgenograms indicated extensive erosion of the petrous pyramid and bony changes in the middle fossa and lesser wing of the sphenoid bone. A crescent-shaped line of calcification outlined the upper border of the tumor. Audiometric and vestibular tests suggested an angle lesion on the right. A large cholesteatoma, measuring 4 by 4 by 7 cm., which had extensively eroded the pyramid of the petrous portion of the temporal bone and occupied much of the middle fossa in front and the cerebellopontine angle behind, was removed surgically. The gross and microscopic appearance was characteristic of an epidermoid. This tumor is one of the largest cholesteatomas reported. Apparently, its origin was in the petrous portion of the temporal bone, which it destroyed; it then spread into the anterior and posterior fossas. The patient made an excellent recovery and was free from symptoms and at work in two months.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

THE ROLE OF BRONCHOSCOPY IN RESUSCITATION OF ASPHYXIA OF THE NEWBORN. H. L. BELL, Eye, Ear, Nose & Throat Month. **31**:601 (Nov.) 1952.

Bell makes a plea for early bronchoscopic examination and aspiration in cases of asphyxia of the newborn. Bronchoscopy should be advocated more universally for infants not responding to conservative therapy and for those beginning to show signs of exhaustion. The trial period of conservative treatment should not be prolonged at the risk of jeopardizing the viability of the cerebral cell structure. Bronchoscopy of the newborn should not be used as a last resort. Passage of the infant bronchoscope is not difficult, and in the hands of a skilled bronchoscopist it is relatively atraumatic.

JENNES, Waterbury, Conn.

RESUSCITATION OF THE HEART IN THE PRACTICE OF OTOLARYNGOLOGY. MERRILL LINEBACK, Laryngoscope **62**:352 (April) 1952.

Cardiac arrest occurred soon after general anesthesia induction preparatory to endoscopy. By quick action on the part of the anesthetist and the surgeon cardiac massage was instituted in time, and complete recovery resulted. There is a discussion of the causes of cardiac arrest and the method of procedure necessary in this emergency.

HITSCHLER, Philadelphia.

RECENT WORK IN VIRUS DISEASES. C. E. VAN ROOYEN, Laryngoscope **62**:805 (Aug.) 1952.

Most viruses can multiply only when in contact with the body cells. The search for antiviral chemicals is made difficult because of this close relationship between virus and cell. It is difficult to affect one without affecting the other. That viruses do not grow in experimental animals also hinders the study of them. There have, however, been advances made in the serological studies of viruses, although the relationship between poliomyelitis and tonsillectomy is not certain. There is no good evidence that inoculations increase the incidence of poliomyelitis.

Discussion of various viral infections is included.

HITSCHLER, Philadelphia.

GRASSES AS FOREIGN BODIES IN THE BRONCHUS AND LUNG. CHEVALIER JACKSON, Laryngoscope **62**:897 (Sept.) 1952.

This is a comprehensive article on grasses found in the bronchi. It is surprising that there are so few instances reported considering the ubiquity of grasses. In general, the grasses are divided into those that lodge and those that travel (one way) downward and penetrate. The reasons for such and the mechanism of the one-way-travel type are discussed, and diagrams are presented showing various kinds of grasses. In addition to the bronchoscopic appearance, diagnosis and treatment are given.

HITSCHLER, Philadelphia.

ODONTOGENIC TUMORS AND CYSTS. WALTER E. HECK, Laryngoscope **62**:1097 (Oct.) 1952.

The understanding of odontogenic tumors and cysts depends upon a knowledge of the embryologic factors concerned. This subject is explained in detail and is well illustrated. The cysts and tumors are classified and their differences explained. Surgical excision is the usual treatment and may have to include the offending tooth or teeth. The invasiveness of adamantinomas is stressed. In this case more radical surgery is necessary.

HITSCHLER, Philadelphia.

TRACHEOTOMY IN BULBAR POLIOMYELITIS. OREL FRIEDMAN and JOSEPH G. GILBERT, Laryngoscope **62**:1112 (Oct.) 1952.

Tracheotomy in bulbar poliomyelitis is done primarily to prevent anoxia and carbon dioxide accumulation due to secretional obstruction of the air passages. It is this anoxia that does more harm to the central nervous system than the virus of the disease.

Indications for tracheotomy are given, together with the advantage gained thereby. Six cases are reported.

HITSCHLER, Philadelphia.

MANAGEMENT OF ACUTE SEVERE HEAD INJURIES. JOSEPH H. SIRIS, New York J. Med. **52**:2642 (Nov. 1) 1952.

Siris says that the majority of head injuries offer no threat to life and little trouble in therapeusis! The period studied is from 1939 through mid-1951. There were 479 patients treated, with 31 deaths—a mortality of 6.5%. Persisting coma is of great importance in all cases. Mortality has been reported to be 70% in the first 24 to 48 hours, a crucial period beyond which chances for recovery improve somewhat. If still conscious after trauma, the patient is in no great danger. The hypothalamus when considered with the frontal cortex has to do with wakefulness, or consciousness. In the assessment of the degree of trauma one must await the

emergence of consciousness. This will be influenced by preexisting intracranial lesions, if that status can be determined, such as intracranial aneurysm, tuberculous meningitis, drug intoxication, cerebral vascular accident, brain tumor, and hysteria. Therefore, a good history is of great importance as to an antecedent condition but may be quite unobtainable. Stupor may be intensified by shock resulting from extracerebral conditions, such as hemothorax or ruptured abdominal organs. Extradural arterial hemorrhage, about 1% of intracranial conditions, practically always requires immediate surgery.

Acute surgical clot should be suspected if there is a unilaterally dilated pupil, focal weakness, Jacksonian convulsions, a post-traumatic lucid period followed by impaired consciousness, persistent impairment of consciousness, or a downhill course. A trivial injury may lacerate a middle meningeal artery on one side, thus leading to grave symptoms. Accessory diagnostic measures are roentgenography of the skull, making of diagnostic burr holes, cerebral aerography, and cerebral angiography. As for angiography, it is more dependable and less formidable than trephination or cerebral aerography.

Many lesions are nonsurgical, and much experience and judgment are required in deciding when or when not to operate. In general, prolonged coma, generalized convulsions, or hemiplegia are not indications for immediate surgical intervention. If "the patient's condition is precarious, it is highly important that unnecessary surgery be avoided."

If status epilepticus persists, the patient's life may be lost. The accompanying stupor may mask an underlying hematoma. "Depressed skull fractures. . . . are not to be regarded as emergent." Brain damage will not be improved by immediate surgery and may be aggravated by the edema incident thereto.

In severe concussion, which is a "derangement of cerebral metabolism," treatment is expectant and consists of management of initial shock; support of the vital body processes through maintenance of optimal fluid; protein, carbohydrate, and electrolyte intake through gavage or parenteral administration of sustenance; control of excessive temperature, and antibiosis. Enhancement of cerebral function may be obtained by oxygenation, sedation, and atropine and general asymptomatic therapy.

The cerebrum is sensitive to lack of glucose and oxygen. Breathing may be obstructed by a back-falling tongue and hypersecretion in nose and mouth. The treatment is obvious, but suction should be emphasized. Occasionally intubation or tracheotomy is in order.

Lumbar puncture, which is too often thought to be imperative, may do more harm than good. It is not unaccompanied by paralyses and other undesirable after-effects in some instances. It "has little to offer. . . and may do harm." Moreover, blood will disappear from the spinal fluid in about five days, tapping or no tapping!

Abstractor's Comment: This is a thoroughly intensive and scientific study of injuries to the head and deserves much more than can be offered in an abstract. There are 27 references to the literature.

I. W. VOORHEES, New York.

NEWER CONCEPTS OF ALLERGY IN EAR, NOSE AND THROAT. MARSHALL H. BARONE, New York J. Med. **52**:2882 (Dec. 1) 1952.

Barone states that treatment of nasal and sinus disease has swung from the surgical to the medical side. In his experience 80% of such patients have some allergy. He quotes G. E. Shambaugh to the effect that from 70 to 90% are allergic. There are three common methods of hyposensitization in pollinosis: preseasonal, coseasonal, and perennial. Injections are begun three months preceding "the hay fever season" and are given weekly. The optimum dose is that one which gives the major immunologic effect. The dilutions are described. A first dose is minimal and is followed by trial doses of 100,000,000 or 10,000,000 or 1,000,000. The latter is rarely used. The spaces are from one to three days according to results. Small oral doses may be given daily during the pollen season and may keep the patient comfortable with or without "the needle." "Children respond very well to this form of coseasonal therapy." The problem of "house dust" is considered but contains nothing new. As for "histamine headache," Barone follows the classification of Hansel, as follows: (1) migraine, (2) atypical migraine, (3) tension headache, (4) histamine cephalgia, (5) atypical histamine cephalgia, and (6) miscellaneous.

The pain is due to vasodilation, not to vasoconstriction, as is often stated. Histamine is given with "good results." Corticotropin (ACTH) and cortisone are useful in some allergies, and J. E. Bordley is cited as seeing nasal polypi almost disappear after two weeks of this nasal therapy! However, the cost, time consumed, and danger involved do not seem commensurate with results ordinarily achieved. A new drug, pyromen® (a pure carbohydrate derivative from certain bacteria), is used in the perennial allergies, with some question as to its efficacy.

Abstractor's Comment: The word cephalgia is used here and is commonly found in the literature today, but the dictionary still holds to cephalalgia.

I. W. VOORHEES, New York.

EAR, NOSE AND THROAT PATHOLOGY IN RELATION TO EYE DISEASE. DARRELL G. VOORHEES, New York J. Med. **52**:2887 (Dec. 1) 1952.

In the department of research of the New York Eye and Ear Infirmary a "chronic infection group" has been set up for the study of focal infections in relation to the eye. The "returns" are not all in as yet, and further studies are planned. Probably otorhinolaryngologists have in the past depended too much on the laboratory and x-ray departments for their conclusions. Voorhees found that both x-ray readings and cultures from sinus washings were deceptive. A sinus may show "clear" after disease has denuded it of mucous membrane. X-ray studies of the ethmoids held first place in confusion. Some which were read "clear" showed only a hollow cuboid at operation because the intercellular septa had been broken down by disease.

As for the washings, "We found pathogenic organisms in sinuses giving clear returns, and no bacteria in cases where gross returns were present following irrigation." A series of 60 antra and 29 sphenoids were studied for kinds of bacteria, with varying types of streptococci and staphylococci evolving; there was no predominant organism. An interesting observation was made as to the adaptation of organisms to changes in environment, especially their hiding away in the deep mucosa and apparent change from aerobic to anaerobic. Iritis, acute and chronic, seems closely related to nasal pathologic conditions.

Diagnosis of "chronic tonsillitis" offered difficulties. One hundred tonsils or tags were studied bacteriologically, but "In only 9 per cent were we unable to isolate organisms that might be considered important. . . . In other words 91 per cent of the tonsils were bacteriologically positive. . . . In my hands preoperative tonsil cultures have not been satisfactory."

I. W. VOORHEES, New York.

PURULENT MENINGITIS IN CHILDREN OF OTO-SINAL ORIGIN. ENRIQUE DELUCCHI, Rev. otorrinolaring. **12**:54 (Sept.) 1952.

Delucchi presents a series of 36 patients with purulent meningitis. Of these, 18 patients showed foci in the ear or the sinuses, while the origin was obscure in 9 patients. Fifteen of his patients recovered. He stresses the fact that in children, in the absence of a typical epidemic, the commonest etiologic factor for purulent meningitis is in either the ear or the sinuses. Secondly, frequently the focus in this area is extremely latent and difficult to determine and evaluate, and secretions may be sterile. He stresses most careful and rigid examination by x-ray, sinus puncture, and surgical exploration and most careful otorhinologic studies, and, finally, when the pathological nature is established in either the sinuses or the mastoid, surgical intervention plus the use of antibiotics is indicated and should be instituted early.

PERSKY, Philadelphia.

CAROTID-CAVERNOUS SINUS FISTULA. B. J. ALPERS, Arq. neuro-psiquiat. **10**:203 (June) 1952.

Alpers reports five cases, one of which was an unusual instance of bilateral aneurysm of the cavernous sinus—a carotid-cavernous sinus fistula on one side and a saccular aneurysm on the other. In three of the five cases the lesion was spontaneous in origin and in two it followed head trauma. In none of the five cases was pulsation of the eyeball found by inspection or palpation. Absence of pulsation is said to be characteristic of lesions of spontaneous origin, but in this series it was absent in the two cases of lesions of traumatic origin. In one case exoph-

thalmos was bilateral. Its occurrence in the opposite eye may be delayed for weeks or months. In four cases there was complete ophthalmoplegia on the affected side, and in one there was impairment only of upward and downward movement. In the first case, in addition to complete ophthalmoplegia on the affected side, there was almost complete paralysis of the extraocular muscles in the other eye, except for slight lateral motion. Visual acuity was reduced in two of the three cases in which it was recorded. There was no optic atrophy or papilledema in this series, though in two cases the disk margins were blurred and in one of these the temporal side of the optic nerve had a grayish discoloration. Arteriography confirmed the presence of a carotid-cavernous sinus fistula in three of the five cases. In one case the condition cleared spontaneously. In four of the five cases the common carotid artery was ligated. In three of the four cases in which follow-up observations could be made the bruit disappeared. In these three cases there was also improvement of the exophthalmos and the movements of the eyeball.

SAVITSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

CHOLESTEATOMAS OF THE CEREBELLO-PONTINE ANGLE. H. OLIVECRONA, Acta psychiat. et neurol. **24**:639, 1949.

In a review of 29 cholesteatomas found by Olivecrona at various sites, 7 in the cerebellopontine angle seemed of special interest because 50% were associated with a typical syndrome. Trigeminal neuralgia involving the third division was frequent in young subjects, of about the age of 3 years. The pain resembled that of the tic douloureux of older persons. Neurologic findings were lacking except in one case, in which the corneal reflex was diminished. No changes were seen in roentgenograms of the skull. The diagnosis was made by encephalography, which usually showed displacement of the cisterna pontis and the cisterna ambiens. The tumor could be removed readily, but the capsule might be adherent to the basilar artery or another vascular structure. Recurrence is considered rare, even if the capsule is not removed. There has been no case of recurrence of angle tumors of this type, even after an observation period of 10 years. In two cases the trigeminal root was partially divided, with little sensory loss, and in another case it was sectioned completely. Tractotomy was performed in one case. The pain subsided in all the cases and did not recur.

PISETSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

A STUDY ON HEREDITARY TENDENCIES OF HARE LIP AND CLEFT PALATE. YUSHI TOGUCHI and KO NAGAI, Otolaryngology (Japan) **24**:240 (May) 1952.

Toguchi and Nagai investigated 156 cases of harelip and cleft palate. There were 83 men and 73 women. There were 77 instances of combined involvement of harelip and cleft palate, 32 cases of lone involvement of harelip, and 47 of cleft palate.

There was definite evidence of consanguinity in these 156 cases. These involved other congenital deformities. There were 10 instances of inguinal hernia, 8 of umbilical hernia, one each of microtia and polyotia, and one of deaf-mutism.

In 24 instances close relatives of the 156 patients showed harelip or cleft palate or both. In 17 instances marriage between consanguineous persons was traced. Other factors that might have influenced the malformation at the time of conception and during gestation were not established.

HARA, Los Angeles.

OBSERVATION ON OPTIC NEURITIS OF NASAL ORIGIN. TSUNEICHI YAMAMOTO, Otolaryngology (Japan) **24**:425 (Oct.) 1952.

Yamamoto records four cases of optic neuritis in which there was a favorable response to retrobulbar injection of 2 cc. of the patient's whole blood. In one patient the procedure was repeated twice and in another three times.

Yamamoto reviews various theories of the origin of optic neuritis and concludes that optic neuritis which disappears without surgical intervention is probably an allergic manifestation. Whether allergy involves the eye or the nose alone or is a systemic reaction is not clear. Further investigation is contemplated.

HARA, Los Angeles.

Directory of Otolaryngological Societies *

INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont. Time: June 14-18, 1953.

Fifth International Congress of Oto-Rhino-Laryngo-Broncho-Esophagology

Place: Amsterdam. Netherlands. Time: June 8-13, 1953.
President: Prof. Dr. Eelco Huizinga.
General Secretary: W. H. Struben, Viottastraat 1, Amsterdam-Z
Scientific Secretaries: Dr. J. Bijtel and Dr. P. G. Gerlings.

International Congress of Audiology

Place: Leyden, Netherlands. Time: June 5-6, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands. Time: June 1-4, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico. Time: January, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3, Pa.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: 1953.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James Milton Robb, 315 Lakeland Ave., Grosse Pointe 30, Mich.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 11-17, 1952.

American Board of Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Roosevelt Hotel, New Orleans. Time: April 21-25, 1953.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Broncho-Esophagological Association

President: Dr. Fred W. Dixon, 1027 Rose Bldg., Cleveland.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Roosevelt Hotel, New Orleans. Time: April 28-29 (afternoons only).

American Laryngological Association

President: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 7.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Roosevelt Hotel, New Orleans. Time: April 26-27, 1953.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis E. LeJeune, 49 Audubon Blvd., New Orleans 18.
President-Elect: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Roosevelt Hotel, New Orleans. Time: April 28-30, 1953 (mornings only).

SECTIONS:

Eastern.—Chairman Dr. Irl H. Blaisdell, 1100 E. Genesee St., Syracuse 10, N. Y.
Place: Hotel Syracuse, Syracuse, N. Y. Time: Jan. 7, 1953.
Southern.—Chairman: Dr. W. W. Wilkerson, Jr., Curtiswood Lane, Nashville 4, Tenn.
Place: Andrew Jackson Hotel, Nashville, Tenn. Time: Jan. 12, 1953.
Middle.—Chairman: Dr. Henry L. Williams, 818 4th St., S. W., Rochester, Minn.
Place: The Drake, Chicago. Time: Jan. 19, 1953.
Western.—Chairman: Dr. Alden H. Miller, 4000 Woking Way, Los Angeles 27.
Place: The Elks Club, Los Angeles. Time: Jan. 24, 1953.

American Otological Society, Inc.

President: Dr. Albert C. Furstenberg, 201 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Roosevelt Hotel, New Orleans. Time: May 1-2, 1953.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Louis Joel Feit, 50 Park Ave., New York 16.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago. Time: Oct. 16, 1953.

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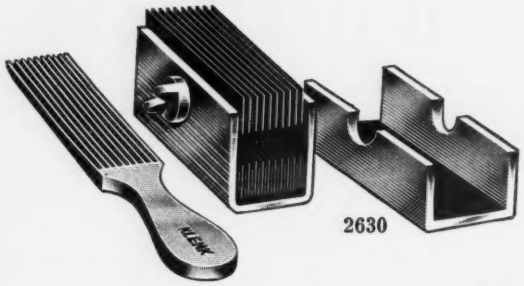

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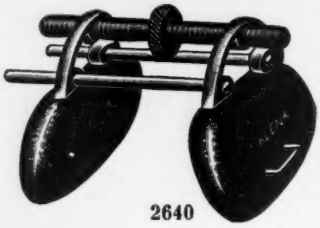

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Archives of Otolaryngology, Sept. 1952: 241-249
The Eye, Ear, Nose & Throat Monthly, April, 1950: 200-203
Bulletin of the Johns Hopkins Hospital, Sept., 1948: 225-228
Archives of Otolaryngology, March, 1947: 294-297

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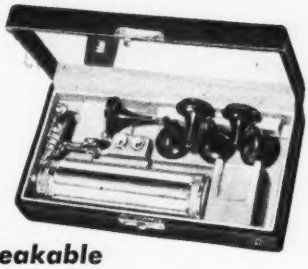

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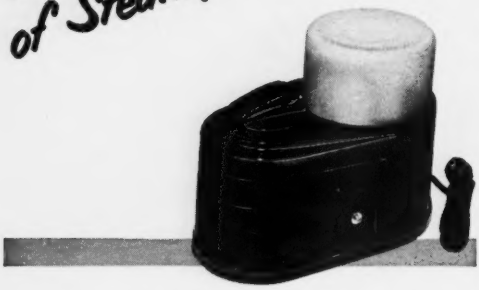

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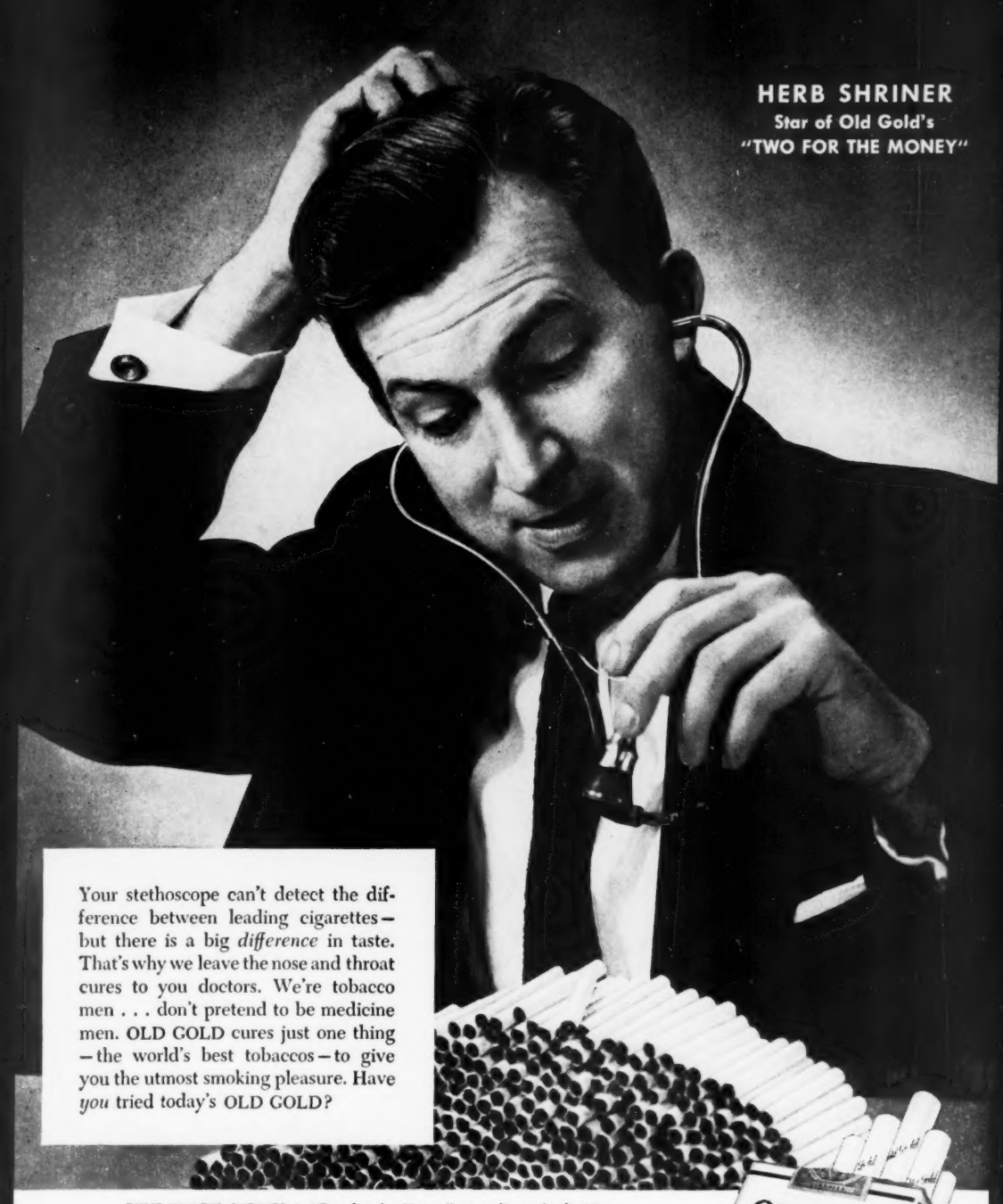

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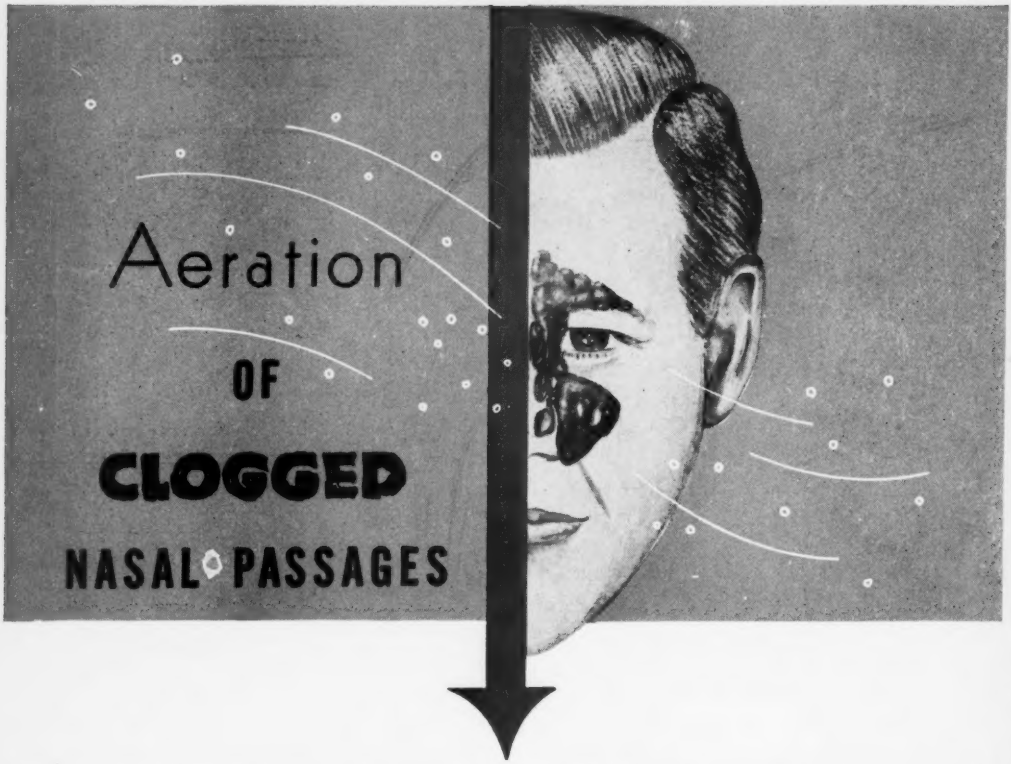

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1. Van Alyea, O. E., and Donnelly, Allen: Arch. Otolaryng., 49:234, Feb., 1949.

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